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MONOGRAPHIC MEDICINE

VOLUME V

DIFFERENTIAL DIAGNOSIS OF INTERNAL DISEASES

BY

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PROFESSOR OF APPLIED THERAPEUTICS AT THE UNIVERSITY OF PENNSYLVANIA;
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TIMOTHY'S HOSPITAL OF PHILADELPHIA, AND TO CHESTNUT
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EDITOR OF TYSON'S PRACTICE OF
MEDICINE, 1913 EDITION

WITH SEVEN COLORED PLATES AND ONE HUNDRED AND NINETY
ILLUSTRATIONS IN TEXT

NEW YORK AND LONDON
D. APPLETON AND COMPANY
1916

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THIS VOLUME IS LOVINGLY DEDICATED TO

MY BELOVED WIFE

SARAH E. FUSSELL

8384

Preface

The author has written the following pages with the hope and expectation that their contents will be of value to the Practitioner of Medicine, as well as to the undergraduate medical student, in separating a certain disease from other conditions with similar symptoms.

That such a Differential Diagnosis is necessary before treatment is undertaken is made evident when one recalls the difficulty that occasionally arises in deciding whether a patient is suffering from pneumonia or whether he is afflicted with appendicitis, or whether the abnormality found in a chest is due to a pleural effusion or to a consolidation of the lung.

The symptoms and physical signs of each of these two sets of conditions may closely resemble each other; their treatment is entirely different; therefore the necessity of an accurate differentiation.

Doctor Barker has written the volumes in this series on Diagnosis dealing with the symptoms of disease, the methods of conducting a complete physical examination and the rules for carrying out the examinations to be made in the laboratory. The methods of conducting these various examinations are given in detail.

This volume presupposes the knowledge and ability on the part of the reader to carry out the necessary procedures. The effort has been made to keep the volume as free from such technical details as possible, in order that the aim of the book—*differentiation*—may always be before the reader.

The author realizes that proper treatment takes chief note of the condition of the patient, whatever may be the name of the disease from which the person is suffering; but he also believes that a recognition of the symptom complex is equally necessary. For instance, it is important to know whether the symptoms are due to tuberculosis or to typhoid fever; this knowledge can be made sure by differentiation. To facilitate this differentiation of diseases, therefore, the following plan has been adopted:

The various groups of diseases are arranged according to the plan adopted by Sir William Osler in his Practice of Medicine. Each disease is then taken up. The symptoms of the disease are first succinctly considered; a list of the diseases which resemble it and with which it may be confused is then given. Each one of these similar diseases is then taken up and their points of difference with the disease under consideration are pointed out. This method of procedure necessitates many repetitions, but it is believed that these very repetitions are of value.

I have presented the subject of Differentiation in as original a manner as possible. Whenever direct quotations or fixed views of others are given, the author has attempted to give due credit.

The section on Diseases of the Nervous System is written by Doctor Charles S. Potts, Professor of Nervous Diseases in the Medicochirurgical College of Philadelphia. The section is founded upon ripe experience.

I am indebted to Doctor James Talley of Philadelphia for the electrocardiograms found in the section on Diseases of the Heart.

Thanks are due to Doctor Robert McCombs and Doctor Robert Pittfield, both of Philadelphia, for revising manuscript and proof, and for many valuable suggestions. My secretary, Miss Alice McGranghan, has given invaluable help.

M. H. Fussell.

Philadelphia.

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DIFFERENTIAL DIAGNOSIS

Differential Diagnosis

Introduction

Diagnosis is a necessity before any rational treatment can be undertaken. Differential Diagnosis is simply the attempt to distinguish a disease of certain characteristics from other diseases which may resemble it either in symptoms or in physical signs.

There are three prime necessities before either a diagnosis or a differentiation of one disease from another can be intelligently carried out. These necessary factors all have reference to the physician attempting to do the work. They are: First, *Knowledge;* second, *Thoroughness;* and third, *Tact.*

Without KNOWLEDGE of the principles of medicine, it is impossible to undertake a diagnosis with any rational expectation of arriving at a proper conclusion. Now that it requires seven years of preparation before one is allowed to practice medicine, it would seem that the preliminary knowledge necessary for a diagnosis is properly taken care of. But the young physician must remember when he leaves his interneship, that while he has a groundwork which should carry him forward in the proper direction, that it *is* only the groundwork, and that his education will never end: that each individual case which he undertakes should be and must be—if he expects to reach the heights—a continuation of his lessons in diagnosis. A man who thus looks upon his patients will daily increase his knowledge and his ability.

THOROUGHNESS is as necessary as knowledge. Given a brilliant man who attempts to shortcut to his goal of diagnosis, and another less brilliant man who is a plodder, who observes, who looks into each detail, the latter man will inevitably make the better diagnoses, and those which will help him most.

Perhaps TACT is as necessary in coming to a final diagnosis as is either knowledge or thoroughness, because unless the physician is enabled to obtain the thorough coöperation of his patient, many points of history, indeed often even points in physical examination, will be concealed from him, and the very points concealed are often those which are necessary for the successful making of a diagnosis.

Important Facts in Diagnosis.—Thoroughness in diagnosis should embrace the entire life history, medical history and a physical examination of every portion of the body plus laboratory methods, to complete the tripod upon which successful diagnosis depends. To be complete, the history of the individual under observation should embrace his sex, nativity, age, social condition, occupation, family history, previous history, and a history of the present condition.

Sex.—It is a well-known fact that certain diseases are present only in males or only in females, and that other diseases are more common in males than in females, and vice versa. It is a fact, of course, that when one is examining a patient, he will know whether the individual is male or female, but if he does not record the sex on his history sheet, then one important point will have been omitted.

Nativity.—The nativity of an individual is of great importance, particularly now that the whole world has become so cosmopolitan. Diseases which were never seen in the temperate zones are now becoming transplanted, or if not transplanted are objects of common experience, and so diseases of the temperate zones are finding their way into the tropics. Amebic dysentery is now quite a common occurrence in temperate climates, while it had its original habitat in the tropics. And so it is with all other diseases which have their beginning in the tropical regions.

Age.—It is necessary to record the age of an individual, because certain diseases are more common in one decade than in another, or have peculiar characteristics in one decade or another, and while perhaps the age limit of diseases is not to be taken as a hard and fast rule, nevertheless it is a most important portion of the history of a case.

Social Condition.—Married individuals frequently have different diseases from those who are single. A genital inflammation in a married woman is very much more likely to be specific than if the same superficial symptoms are present in a single woman. The history of numerous miscarriages is of value often in determining whether or not a case is luetic.

Occupation.—The occupation of an individual is of importance, certain occupations being subject to special diseases. Lead workers, for instance, are affected with lead poisoning very much more frequently than are others.

Family History.—The family history is of importance, for while it is a fact that the effect of heredity as to the causation of diseases is now less perhaps than was previously thought, a knowledge as to whether an individual has had a number of cases of tuberculosis in the family or of syphilis in the family is of vast importance in considering the symptoms of the patient in relation to these particular diseases.

Previous History.—The history of previous diseases is of the utmost importance. It is much more likely that a person who has suffered at some previous date from pulmonary hemorrhages is affected with tuberculosis, than an individual who has never had a pulmonary hemorrhage. Another individual who has had frequent attacks of anemia, of weakness, of breathlessness, and who is anemic at the time of examination, is much more likely to be suffering from progressive pernicious anemia than an individual who has no such history.

Present History.—The history of the present condition, of course, is all-important, and should be obtained in a routine manner. It is well at first to endeavor to avoid leading questions, because there are certain classes of individuals who answer affirmatively or negatively, as the case may be, simply because the question is given a prominent place. It is wise to begin the questioning of the present condition by asking distinctly how long it has been since the individual was in perfect health, and follow this question by asking him how he was first affected, and then lead him up to his symptoms at the present time. After he has given this somewhat voluntary history, ask categorically specific questions as to symptoms, relating to each of the systems: the digestive system, respiratory system, circulatory system, urinary system, nervous system, and the locomotor system. In this way one obtains a history upon which he can depend.

Recording the History.—It is not wise, either, that this history should simply be taken and not recorded. Every physician, for his own sake and for the sake of his patients, should record in some manner a history taken after the style I have suggested. The method of recording a history must depend entirely upon the person who takes it. Perhaps the best way is to take notes at the bedside, and then have them transcribed. This is entirely impracticable, however, for an individual who does not have a secretary, and who is hurried from one place to another, but it is entirely practicable to carry in one's wallet or handbag cards upon which a history can be written. These cards may be of a convenient size—the size of one's prescription blanks. After taking the history of one or more cards, he then should make at once a cross index. If the individual is suffering from pneumonia, or appears to be suffering from pneumonia, write at the head of another card of the same size as the one on which the history was taken the word *Pneumonia*, and under the word Pneumonia the name of the patient. When the physician reaches home these cards can be at once indexed in separate boxes under the proper letters, taking but a few seconds of time (the history card to be indexed according to the name of the patient—the cross index under the name of the diseases). If this is conscientiously done, the patient will be better off because a record will be kept of the condition as it progresses; the physician will be better off because he is teaching himself methods of routine observation, and because he will have data upon which to make observations.

Physical Examination.—Every physical examination must be done in a routine manner, and notes made of the condition of the various parts of the body, whether those parts are in a normal or abnormal state. Nothing is more disappointing than to refer to one's notes, and to discover that the condition of the eyes, or of the reflexes or of the heart, for instance, is not mentioned. There should be a positive or a negative note made about every single organ. First, the body should be inspected, and notes made as to the general condition of the head, the trunk and the limbs; as to the absence or presence of enlarged glands; as to the absence or presence of discoloration of the skin, and the appearance of any abnormality which comes to one's observation. Then the condition of the eyes should be noted; the condition of the face, the ears, mouth, teeth, the throat, the neck; the cardiac dullness, the rhythm of the heart, the position of the apex beat, the character of the sounds of the heart, the presence or the absence of cardiac murmurs; the presence or absence of abnormal areas of dullness in the chest; the condition of the lungs, whether there is dullness, whether the breath sounds and voice sounds are normal; the area of liver dullness; the area of splenic dullness; the area of stomach tympany, whether there are areas of tenderness anywhere in the abdomen, whether there is distention of the abdomen, whether a tumor mass can be felt or not; the condition of the deep reflexes; indeed not a single organ or part of the body must escape observation. A physical examination should always be made with the portion of the body being examined stripped. No one can possibly give a proper idea of the condition of the heart, the lungs, the abdomen or the limbs if these parts are covered with clothing.

Laboratory Methods.—Laboratory methods have become such an important part of diagnosis that one cannot afford to neglect them in any case. To do so is to court error. In order to attend to the laboratory side of the case, a physician must either know how to make laboratory examinations and practice them, or he must have them done for him by an immediate assistant or at some laboratory. Certainly at this age, no physician can afford to be unable to interpret any laboratory finding which is sent to him. It is not a question of expense; it is a question of necessity. If an individual cannot afford to pay for laboratory work, and if a physician feels that he cannot afford to do it without pay, then that patient should be sent to some proper institution where the work can be done gratis, and where the individual can have proper attention. If every practicing physician carried out these rules, there would be less fault-finding on the part of patients, and what fault-finding occurred would have very little basis. The patient would receive benefit, and the physician would be doing work worth while. Laboratory examinations which are absolutely necessary are:

Urine Examination.—A routine urine examination must be made in every single case that comes under our observation. This is a necessity. The presence of albumin, as is well known, does not always mean that an individual has nephritis; the presence of pus in the urine does not always mean that an individual has cystitis or pyelitis, but it does mean that there is something wrong with the genito-urinary tract, and that something cannot be diagnosed and will escape observation unless the urine of every patient be examined.

Blood Examination.—If possible there should be a complete blood count in every case. There are some conditions in which this is not a necessity, but the more often we make a routine blood examination, that much less likely are we to make mistakes. It is manifestly impossible to make a diagnosis of one of the anemias, including leukemia, without a blood examination. A patient often appears pale when he is really not anemic, and it is futile, without knowing the hemoglobin and the red cell count, to apply treatment to such an individual. The various complement fixation tests are imperatively necessary in helping to form an opinion as to the condition in certain cases. Blood pressure, both the systolic and diastolic, should be taken as a routine matter. It is true that we do not yet know how to interpret the findings of every case, but it is of the highest importance that we should know whether an individual has high blood pressure or low blood pressure, or whether it is normal.

Examination of Stomach Contents.—The stomach contents should be examined in all cases of gastro-intestinal disease. While it is true that we are beginning to place less confidence and to act less surely upon the gastro-intestinal findings, yet it is a fact that they give us much useful information, and point the proper way to treatment in many conditions.

Examination of Feces.—An examination of the feces for ova, for occult blood, for fat, and for undigested meat fibers is equally necessary.

X-ray Examinations.—X-ray examinations are of the greatest value in all diseases of the bone, whether traumatic or not; in conditions of the chest—particularly where some sort of an effusion is considered; they are an important help in the diagnosis of tuberculosis, and are of the greatest amount of value in examination of the digestive organs.

Examination of Cultures from Throat.—In general practice, examinations of cultures from the throat are of extreme importance. As will be stated later on, it is utterly impossible, in a certain number of cases, to make a differential diagnosis between diphtheria and follicular tonsillitis without a throat culture. A culture is easily obtained, is safely done, and saves numerous errors.

Conclusion.—I can no more fittingly close this appeal for thorough examination with the aid of laboratory methods than by quoting from Osler's "Modern Medicine," First Edition, where in the Introduction he says:

"Just as the clinical laboratory is a necessity to the hospital physician engaged in the solution of the most advanced problems in medicine, so the private laboratory is indispensable in the every-day work of the busy practitioner. Urine analysis, blood counts, sputum examinations, chemical analysis of stomach contents, all these should be done at home; at first, by the physician himself while not too busy, later by an assistant. This may seem to be asking a great deal in the heavy routine of the day, but it is not asking too much, and it will be done more and more when we send out our students familiar by long practice with the use of the microscope and other instruments of precision. It makes the practice of medicine of absorbing interest when one feels he is approaching the study of a case equipped with modern methods, and it is the neglect of these accessories that makes so many men fall into slipshod habits of diagnosis, and still more careless methods of treatment. Asked the single most powerful weapon today in the hands of the profession against quackery of all sorts, I would answer: 'the little laboratory room attached to the office of the general practitioner.' Nor is it asking the impossible. I know many busy men who utilize to the full all these resources of our art."

Section I

Specific Infectious Diseases

A. Bacterial Diseases

1. Typhoid Fever

Origin.—Typhoid fever is an acute transmissible disease, due to implantation of the Eberth bacillus (Bacillus typhosis). Cultures of this bacillus may be obtained from the blood, from the eruption, from the urine, from the stools, and from the spleen.

Onset.—Usually typhoid fever begins with malaise, headache, symptoms of indigestion, tendency to diarrhea, and sometimes nosebleed lasting one week to ten days. Occasionally, however, the onset is sudden and there are no prodromal symptoms, the patient being critically ill within a few hours with high fever, headache, delirium and abdominal symptoms —presently to be described. This feature was present during an epidemic in Roxborough, Philadelphia, the patients all being observed at St. Timothy's Hospital. Occasionally the onset resembles closely an attack of meningitis with delirium, increased reflexes, and opisthotonos. A spinal puncture should invariably be made in all such cases. The fluid will be clear or cloudy; it may contain typhoid bacilli, pneumococci, tubercle bacilli, or meningococci, and by this finding the differential diagnosis between these conditions can be made.

Various Forms.—(1) Gastro-intestinal Form.—Sometimes the gastro-intestinal symptoms are the prominent ones which attract the attention of the physician, and the case is mistaken for one of gastro-enteritis, but the persistent fever and the presence of a Widal reaction marks the case as one of typhoid fever.

(2) Pulmonary Form.—Again acute pulmonary symptoms are present in the beginning which so dominate the case that the attack is mistaken either for acute bronchitis or pneumonia, but the aberrant physical signs, the absence of leukocytosis, the persistent fever, and later the characteristic blood reaction, again make the case one of typhoid fever.

(3) Renal Form.—As will be discussed presently there are certain cases which in the onset are dominated by the presence of an acute nephritis, the latter even hemorrhagic in form. Such a case differs in many particulars from a simple acute nephritis especially in the continuation of the fever.

(4) Follicular Tonsillitis.—Follicular tonsillitis in one case of the author's was such a prominent symptom that the case was looked upon as a simple case, until with the disappearance of the symptoms of sore throat, the persistence of the fever brought about the proper diagnosis.

(5) Ambulatory Form (Walking Typhoid Fever).—Again the symptoms may be so mild that the patient does not believe himself seriously ill until he is brought to the realization of his condition by the sudden appearance of serious symptoms or of some complication.

(6) Abortive Typhoid Fever.—There exist certain attacks in which the case begins with all of the characteristics of an ordinary case of typhoid fever, but in which in the course of a week or ten days the temperature is normal and the patient convalescent. These cases can only be surely diagnosed by the presence of rose spots, enlarged spleen and the Widal reaction or blood culture, and perhaps the latter two are absolute necessities for a positive diagnosis.

(7) Afebrile Forms.—Afebrile forms are reported in which the individual has many of the symptoms of typhoid infection without fever. Here only a blood culture will make the diagnosis. Even the presence of a Widal reaction is not sufficient evidence of typhoid fever in these cases because of a possibility of the Widal reaction being caused by a previous attack of typhoid fever or by vaccination against typhoid fever.

Symptoms.—Temperature.—When the usual case is seen early, the morning temperature is only slightly above normal, the evening temperature may reach 100° F. The temperature rises gradually during the first four days to one week. At the end of the week the temperature rises to 102° F. in the morning, with an evening rise to 103° F. or over. This fastigium is maintained for about two weeks, when the temperature begins to fall first in the morning, gradually in the evening. At the end of four weeks the temperature is normal night and morning. This is what may be called the "textbook" range of typhoid fever (Fig. 1). There are many variations. The rise may be abrupt to the fastigium, it may be unusually slow. During the height of the temperature there are occasional abrupt falls not due to hemorrhage, and not due, so far as can be discovered, to the result of treatment (Fig. 2). This abrupt fall, especially when it comes late in the disease, is most confusing, because of its likeness to the temperature drop often observed during a severe hemorrhage. There is this difference however. The temperature drop due to a hemorrhage is almost without exception accompanied by a rapid pulse and increase in respiratory rate. The pulse and respiratory rate are not

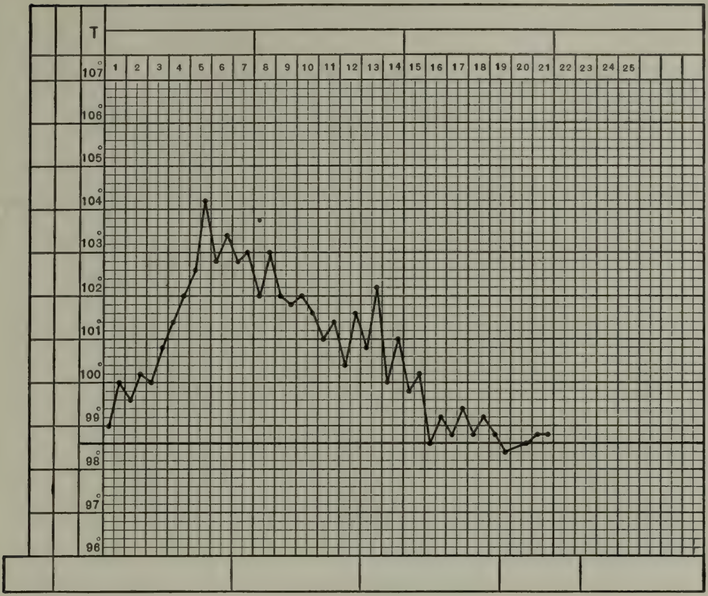

Fig. 1.—Temperature Chart of Mild Uncomplicated Typhoid Fever.
(Personal Observation.)

increased in the drop due to unimportant and unknown conditions. Frequently in the third week there is a greater difference between the highest

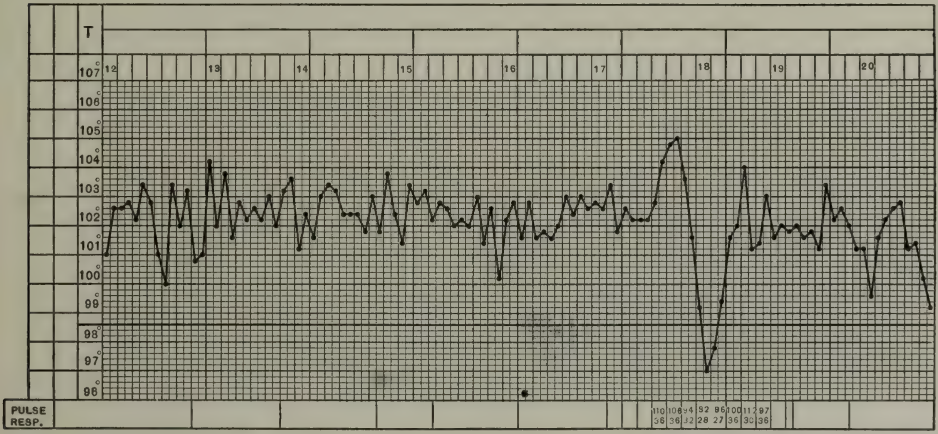

Fig. 2.—Abrupt Fall in Temperature during Typhoid Fever Not Due to Hemorrhage.
(Personal Observation.)

and lowest temperature during the day, the range in temperature becoming more remittent in type.

During convalescence there is occasionally an unusually prolonged intermittent fever; this is at times doubtless due to nutritional fault and

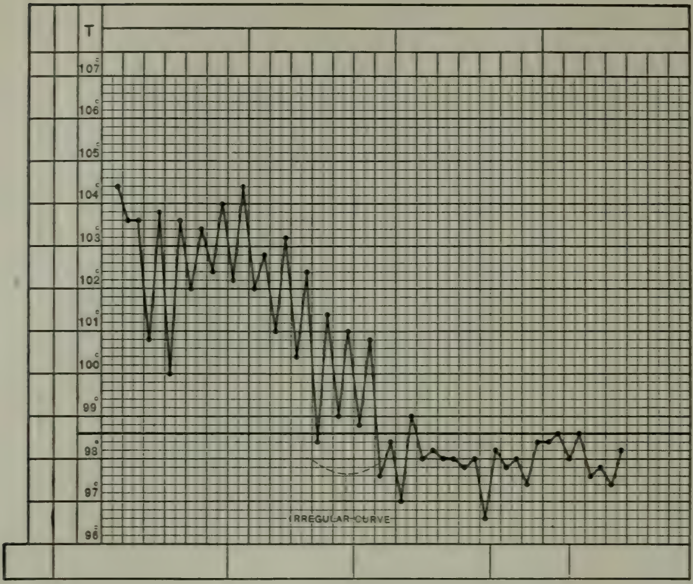

Fig. 3.—Intermittent Temperature Curve Seen at End of Typhoid Fever. (Personal Observation.)

may then be promptly ended by careful feeding (Fig. 3). It is less likely to occur when patients are liberally fed. Sometimes it appears to be due to a true septicemia.

A relapse in typhoid fever occurs in a certain proportion of cases, and is indicated by a gradual rise of temperature, with a recurrence of all or most of the original symptoms of the disease. The temperature lasts for a week or more, and then falls gradually, just as it did in the first attack (Fig. 4).

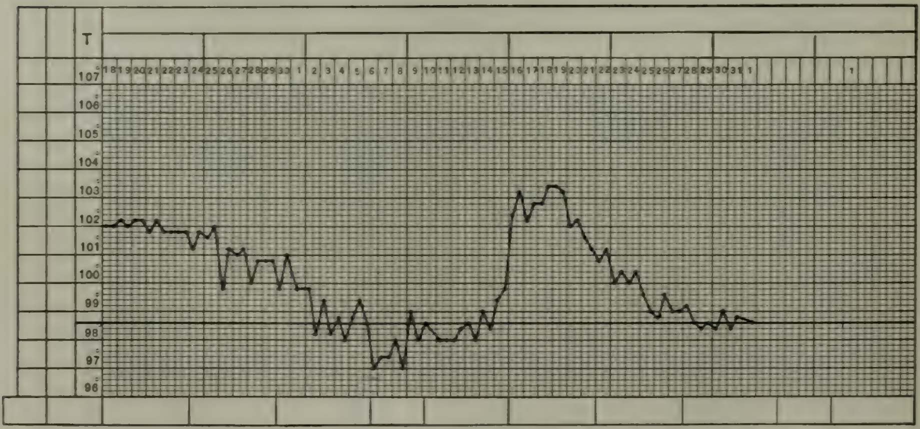

Fig. 4.—Chart Showing Relapse in Typhoid Fever. (Personal Observation.)

A recrudescence occurs when a case which has been running a normal course toward convalescence—but where the temperature has not reached the normal line—gradually begins to have an increased severity of the symptoms, together with a return of the temperature to a higher level (Fig. 5).

Occasionally there is a sudden or gradual rise of temperature lasting from a few hours to days. This may be due to an increased toxemia, or to some local inflammatory condition. If the latter is the case there is almost without exception an accompanying leukocytosis.

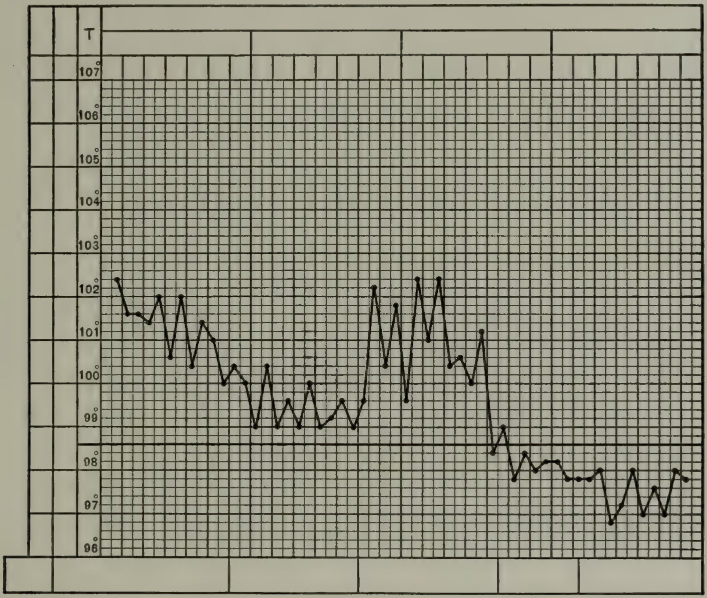

Fig. 5.—Chart of Recrudescence in Typhoid Fever. (Personal Observation.)

Nervous System.—*Headache and Delirium.*—Headache is often the initial symptom. It may be mild or severe. During the first week or ten days it is very frequently unbearable. Delirium is fairly constant, varying from mild mutterings during sleep or when first waking to wild sleepless mania demanding opiates for its control. It was present in 24 out of 48 cases in St. Timothy's Hospital. Instead of the delirium being maniacal, it is still more frequently low and muttering in type. The patient is unconscious, or realizes his surroundings only upon being awakened and his attention being held by effort. The face is flushed, the tongue is dry, the teeth are covered by sordes (removal of the sordes and cleansing of the mouth will frequently improve the condition).

Carphologia, twitching of the arms and legs, involuntary defecation and urination (the typical typhoid state) frequently supervene. A case may begin in the usual way and gradually develop meningeal symptoms, similar in respect to those in the early meningeal forms. There is more of a tetanic form to the muscular disturbance, however, in these cases. The muscles are more continuously tense. If the patient has been taking strychnin in large doses, the drug must at once be stopped, as an aid to diagnosis if for no other reason, for the cases have a marked resemblance to strychnin poisoning in mild form. Here the recourse to spinal puncture should be made to be certain whether one is dealing with a true meningitis.

Meningitis.—A true meningitis occurs as the direct result of the typhoid bacillus or as the result of an actual purulent meningitis. In Cole's cases the typhoid bacillus meningitis was accompanied by a clear spinal fluid, while the purulent cases had a cloudy fluid.

Psychoses.—Actual psychoses occur sometimes in the beginning, but more frequently during the convalescence. Some of the cases develop an insanity during convalescence which is apparently a continuation of an early delirium, but frequently the insanity occurs first during the convalescence. It may be so prolonged as to require institutional treatment.

Tremors.—Tremors are common. They may occur during the height of the fever, or during convalescence. Its degree appears to depend upon the systemic disturbance due to the toxin of the disease.

Neuritis.—A neuritis often occurs either general or local. Tender toes, the result of a local neuritis, is quite a common symptom. Frequently the tenderness is so great that the bed clothing has to be raised from the feet by means of some support.

Digestive System.—*Condition of Tongue.*—The tongue is in the beginning rather pointed, with a red tip, and covered with a thick whitish fur. This condition rapidly changes to dryness, when the whole tongue is chiplike in dryness and is covered with a thick brown fur. As the condition of the patient improves the tongue again becomes moist and gradually improves during convalescence. In mild cases the tongue may be very little coated and may never become dry.

Vomiting.—Vomiting rarely occurs, but in severe toxemia, or if the stomach is overloaded with food, there may be vomiting.

Bowel Movements.—Diarrhea is the rule. The bowel movements are usually loose and ill-smelling. Instead of diarrhea, constipation may be present from the beginning to the end of the disease.

Tympanites.—Tympanites, frequently the result of intestinal paresis, may amount to monstrous distention, at times so great as to displace the heart and liver upwards, and it may even obscure part of the liver dullness.

Intestinal Hemorrhage.—Intestinal hemorrhage may occur as the result of sloughing of an intestinal ulcer. It can be discovered by the presence of blood in the stool. Hemorrhage is greatest and most common from the intestines, though it may occur from the stomach and from the nose.

The author saw one case of death directly attributable to epistaxis, and another case of epistaxis in which the fall of temperature was as great as any fall seen in hemorrhage from the bowel.

Intestinal hemorrhage may be slight or severe, it may be a single hemorrhage, or may be repeated many times. The intervals of the hemorrhage may vary from a few hours to a number of days. Very slight hemorrhage gives no symptoms save the presence of blood in the stool.

Grave hemorrhages are indicated by rapid pulse, pallor, and drop of temperature sometimes before the blood appears in the stool. There is also likely to be a fall in the blood pressure and in the percentage of hemoglobin, and in the number of red cells. After a hemorrhage there is usually a leukocytosis. This may go as high as 20,000 or 25,000 leukocytes per cm. On the other hand there may be no leukocytosis. If symptoms of hemorrhage occur and no blood appears in the stool, dullness may be elicited in the right flank, due to the collection of blood in the cecum. Hemorrhage occurs most frequently in the third week of the disease.

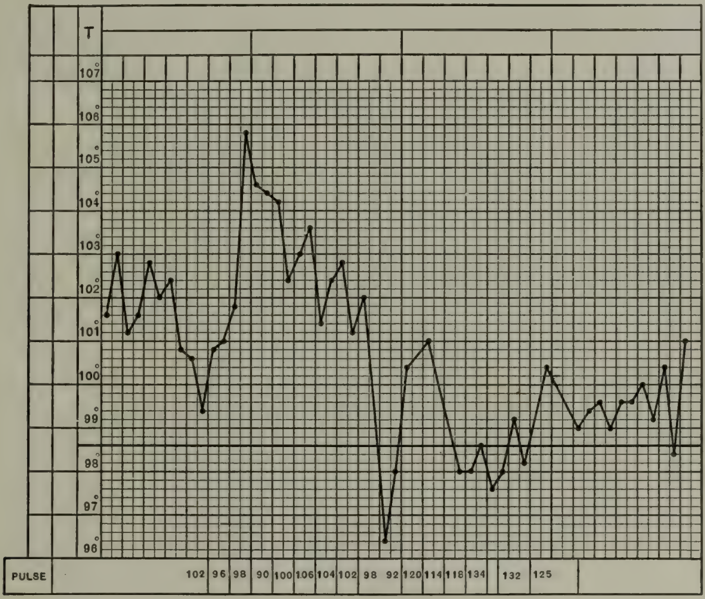

Fig. 6.—Chart Showing Drop of Temperature Due to Intestinal Hemorrhage. (Personal Observation.)

Perforation of Bowels.—Perforation of the bowels occurs in a certain number of cases; in a series of 5,168 cases collected by the author, it ranged from 1.36 per cent in 733 cases in free diet to 2.38 per cent in 4,654 cases on liquid diet. It occurs most frequently in the third week, as does hemorrhage from the bowels.

Abdominal Pain, etc.—It may be the first symptom to attract serious attention in ambulatory cases as it was in two cases of the author's at St. Mary's Hospital. The usual symptoms are *sudden severe pain,* with *local tenderness* and *local resistance,* most common in the right lower quadrant of the abdomen. The symptoms, especially if accompanied by a rising leukocytosis, give sufficient reason to call a surgeon at once for the purpose of diagnosis and subsequent treatment. The pain is usually paroxysmal, though it may be constant, and when it is paroxysmal the intervals of relief are quite short. There is often no rise in pulse rate, and no fall in temperature. To wait for these is to insure disaster.

We must not wait for disappearance of liver dullness, great distention of the abdomen, and disappearance of peristalsis, or rapid running pulse with hippocratic facies. These are signs of general peritonitis. Usually the bowels do not move after a perforation, but the author has seen a copious bowel movement after the occurrence of a perforation proven by operation.

If suspicious symptoms continue and cannot be explained on other grounds than perforation, an exploratory operation should be done.

GALL-BLADDER.—*Inflammation of the Gall-bladder.*—This often occurs with pain and tenderness in the region of that viscus. It may result in nothing but local tenderness, but on the other hand it may continue to suppuration and demand operative interference.

SPLEEN.—The spleen enlarges about the end of the first week. This enlargement may be diagnosticated by increase in dullness in the splenic area and palpation of the spleen below the edge of the ribs. The following is the most certain method of discovering the enlargement: the patient lies on his back with the legs well drawn up, and is requested to take a long breath; with the fingers of the examiner gently but firmly depressing the abdominal wall at the margin of the ribs, the edge of the spleen can usually be felt when the organ is enlarged. Certain individuals, notably soldiers, retract the abdomen when requested to take a long breath. This defeats the object of the test, and the patient must be instructed to inspire by the use of the diaphragm, which will immediately lower the spleen.

RESPIRATORY SYSTEM.—*Bronchitis.*—Bronchitis is the rule. It is a rare case which goes through an attack without more or less cough; râles can practically always be heard upon auscultation.

Laryngitis.—Laryngitis is frequent. This may be a mere catarrhal laryngitis, or it may be a true ulceration eventuating in a perichondritis most frequently present in the thyroid cartilage. Because of the occasional occurrence of this perichondritis great watchfulness must be exercised whenever a hoarseness presents itself.

Pneumonia.—The French have described a form of typhoid fever with marked pulmonary symptoms, often an early pneumonia. Here the pneumonia often marks the beginning of typhoid fever. The usual blood examination, and finding typhoid bacilli in the sputum, will frequently clear up the case.

Ear.—*Otitis media.*—Otitis media frequently occurs. The ears should be carefully watched, especially when the patient is unconscious, in order that this complication may be observed and treated.

Kidney.—*Albuminuria and Casts.*—Practically all severe cases of typhoid fever have more or less severe albuminuria; occasionally there are tube casts. This is simply a toxic albuminuria of the same character which may occur in any severe septic condition.

Nephritis.—More commonly than is usually suspected, there is a true severe nephritis, which becomes a threatening element. For this reason a routine urine examination, both chemical and microscopical, should be the unvarying rule in every case of typhoid fever. Typhoid bacilli are present in a large percentage of the urines examined. Diazo reaction should always be made use of, not because it is pathognomonic of typhoid fever, but because its persistent absence argues against typhoid, and its reappearance in the urine shows a probable relapse of typhoid, rather than the occurrence of some complication. Occasionally the case begins with nephritis as the initial symptom, even a hemorrhagic nephritis—the so-called renal form of typhoid fever. This form described by Didon occurs most constantly in the second week of the fever.

The Heart.—The heart suffers from the general toxemia. There is an increased number of beats, the rate ranging from 80 to 110. In severe cases the pulse may become running and dicrotic. A true myocarditis may occur, leaving its effects long after the recovery of the patient. A hemic murmur is common, though a true endocarditis rarely occurs. Pericarditis may be an accompaniment, but is rare.

Blood.—*Widal Reaction.*—The agglutination reaction made known by Widal is present. According to Cabot, in a series of 5,078 cases 97.2 per cent exhibited it and in a large percentage of the cases before the eighth day. It appears about the sixth or seventh day of the disease, though it may be delayed until late in convalescence.

Leukopenia—Leukocytosis.—Leukopenia is the rule. However in many cases of typhoid fever there is a slight increase of the leukocytes in the beginning of the disease, but on the third or fourth day the leukocytes will be found at or below normal.

The differential count, which is of great importance, will show an increased number of lymphocytes in uncomplicated cases. Leukocytosis may occur, often after hemorrhage, or perforation. In the latter case counts must be made every hour to be of any value. If a continuous leukocytosis occurs, either the case is not typhoid, or some complication is occurring in the course of typhoid fever. A blood culture made in the first two weeks will show the presence of typhoid bacilli in the blood in a certain proportion of cases.

SKIN.—*Rash.*—A papulomacular rose colored rash appears on the seventh or eighth day, varying in degree from a few scattered spots on the abdomen, to an abundant eruption over the entire body. This rash disappears on pressure and reappears almost instantly upon removal of the pressure.

Pressure may be made either by firmly pressing the spot itself with a finger and quickly removing it, or by influencing the site of the spot by putting one finger on each side of the macule and drawing them apart, and then quickly removing the fingers. The spot will completely disappear when the pressure is applied, to reappear instantly on removing the pressure. The rash appears in *crops*, which is quite as distinctive as the character of the individual spot. This rash should be sought for carefully; if none be found on the abdomen, other parts of the body must be searched.

Furunculosis.—Furunculosis is common. When it is present it often proves a severe complication. It is apt to occur in the late stages of the fever or in convalescence. Pure cultures of typhoid bacilli have been found here in some cases, though staphylococci are more commonly the infecting organism.

Bedsores.—Bedsores are common. In the latter stages of severe cases of the disease the vitality of the individual is so lowered that the least pressure causes deep sloughs over the parts touching the bed, and a water bed or an air bed becomes a necessity.

Diseases to be Differentiated from Typhoid

Typhoid may be differentiated from:

Tuberculosis
Malarial fever
Pneumonia
Endocarditis
Typhus fever
Appendicitis and other abdominal inflammations
Paratyphoid and other colon bacilli infections
La grippe
Trichinosis
Osteomyelitis
Septicemia
Measles and other exanthemata
Hodgkin's disease
Meningitis
Syphilis
Acute nephritis.

Indeed, it must be differentiated from any disease which has fever as a symptom, in which the pathognomonic signs of the condition are delayed for any reason.

TUBERCULOSIS.

Tuberculosis with its insidious beginning, often when the lungs are slightly affected, or not at all, is frequently mistaken for typhoid fever. In the mild cases of tuberculosis of the lungs the fever is not so regular nor does the temperature run so high, and the patient is noticeably so little ill that he constantly begs to be up and about. Repeated physical examination will frequently reveal a small area of consolidated lung, usually at the apex, possibly at the base or in the interscapular space. The whole chest—bared—must be repeatedly examined to avoid missing the diseased area. A little dullness, or a spot with prolonged expiration, a few moist râles confined always to one spot, may give the necessary diagnostic point. Glandular tuberculosis may be made out by the presence of a bunch of enlarged glands in the neck or axilla, or dullness along the bronchus anteriorly, will show the true nature of the disease.

The absence of the Widal reaction is always suggestive of some other fever than typhoid.

Even with the smallest amount of sputum, tubercle bacilli may be found, and the search for them should never be neglected. The tuberculin reaction after the method of von Pirquet is of value in very young children, and often will give the deciding point.

Miliary tuberculosis, and in the beginning tubercular meningitis, are the two conditions which are perhaps most frequently mistaken for typhoid fever.

In both of these conditions there is leukopenia, just as there is in typhoid fever. The persistent absence of the Widal reaction is of great value in separating miliary tuberculosis and tubercular meningitis, for, as stated above, the Widal reaction was present in over 97 per cent of the cases of typhoid fever observed at St. Timothy's Hospital. When the case is one of tubercular meningitis, the presence of tubercle bacilli in the spinal fluid will be positively against typhoid fever, and will enable a positive diagnosis of tuberculosis to be made.

Miliary tuberculosis may be suspected when there is a fever of a continuous or slightly remittent type, with no Widal reaction, with a leukopenia, and lymphocytosis.

MALARIAL FEVER.

Malarial fever is an *intermittent fever*—which point, above all, should be of decisive value. The presence of the plasmodium malariae in the blood—and every general practitioner should be able to recognize this organism—of course will be proof positive of the presence of malaria. However there are a few isolated cases in which there is a mixed infection, the patient suffering from both malarial fever and typhoid fever.

Under these circumstances the Widal reaction must be sought for, as well as the other signs of typhoid, such as spots, enlarged spleen, etc. There can be no valid excuse, particularly in the temperate climates, of confusing the two diseases.

The autumnal form of malarial fever with its lack of intermittent form of temperature, the persistence of a remittent but more or less continuous fever, and the scarcity of the plasmodium in the circulating blood, may give rise to real difficulty in diagnosis. Where there is difficulty, the blood should be examined two or three times daily, and the therapeutic test of quinin should be used. Any fever which resists for several days the action of large doses of quinin is not malarial. Sometimes the splenic enlargement, even in acute malaria, is greater than in typhoid fever.

Pneumonia.

Pneumonia is occasionally so lacking in physical signs in the chest, that it is a matter of concern to differentiate it from typhoid fever. Under these conditions the most valuable physical sign is examination of the blood. Pneumonia will in the vast majority of cases give a leukocytosis, varying from 12,000 to 30,000 white cells per cm. Polymorphonuclear increase, hurried breathing, and developing physical signs in the lungs will complete the diagnosis. Herpes usually about the face is notoriously absent in the majority of cases of typhoid, and when present in doubtful cases, is a valuable sign of some infection other than typhoid fever.

Endocarditis.

Endocarditis is constantly mistaken in the early stages for typhoid fever. Here the rapid anemia in malignant cases, the irregular septic type of fever, the developing heart murmurs are signs which point the way to a proper diagnosis. The cardiac murmur in endocarditis is one of the earliest signs. It is late and it is soft in cases of hemic murmur in typhoid fever.

The blood examination is of value. There is without exception a polymorphonuclear leukocytosis, and in the great majority of cases a blood culture will show the presence of the infecting organism in the blood of endocarditis (See Fig. 7, page 19).

Typhus Fever.

Until the time of Louis, typhus fever and typhoid fever had been confounded. With our present knowledge of the pathology of the two conditions, we know they are different diseases. Usually there is a history of exposure to some other cases of typhus. If there is an epidemic of typhus fever the chances are the supposed infection is due to typhus fever. The onset in typhus is abrupt. The rash which is petechial in character and does not disappear on pressure, occurs all over the body on the third day. If there has been exposure to overcrowding under filthy conditions, there is still more cause for the diagnosis of typhus. The course of the fever, whether ending in recovery or death, is shorter in typhus fever.

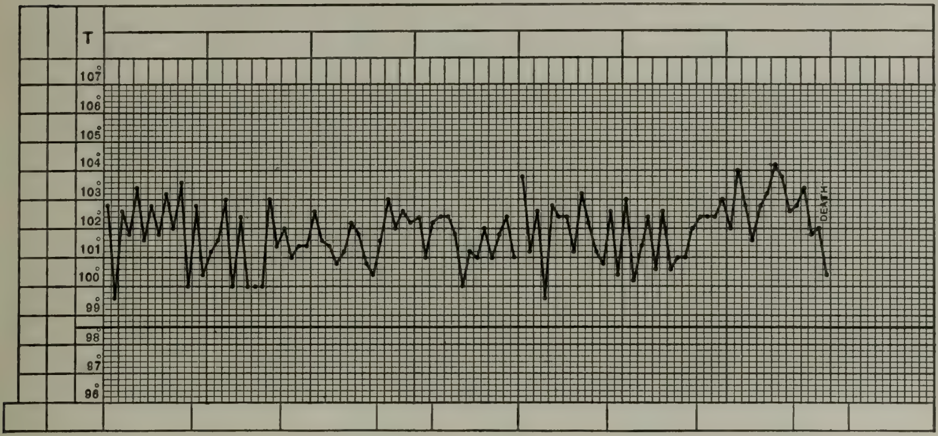

Fig. 7.—Temperature Chart of Malignant Endocarditis. (Personal Observation.)

The discovered fact that typhus fever is transmitted by the bite of the body louse is of some value. Of course typhoid infected people may be filthy, but the discovery of the body louse on the body of an individual sick with a disease of sudden onset, is of some value, especially when that individual belongs to the vagrant class.

Much difficulty may be found in separating the mild cases of typhus from typhoid fever. The absence of leukopenia, the abruptness of the onset and short duration of mild typhus fever, and the absence of the Widal reaction will help much to make the diagnosis. Again, sporadic cases of typhus are apt to be followed by others of the same character if the surroundings be unsanitary. And lastly, Brill's disease, by which name mild cases of sporadic typhus fever are known, may be communicated to monkeys by inoculation of the monkey with the blood of the sick individual. This valuable laboratory method of diagnosis can be made use of in cases of fever with signs of typhoid, which lack the Widal reaction and the blood cultures which are characteristic of typhoid or paratyphoid fevers. A complement fixation test is now used which is thought to give positive evidence of typhus fever.

Appendicitis.

Appendicitis may be mistaken for typhoid fever, and vice versa. Years ago, before appendicitis was so readily recognized, the mistake was much more common. Even now diagnostic acumen is necessary in order to separate the cases in certain instances.

Appendicitis on careful examination will give tenderness and resistance over the appendiceal region. Leukocytosis can be discovered. The previous history may not be unlike in the two conditions.

With certain cases of appendicitis fever is the main symptom, but temperature taken every three hours will show the fever of appendicitis is septic in type, that there is a marked difference in degree between the morning and evening temperature, while there is much less difference in degree in the temperature in typhoid fever. Again, regarding the variations in degree, particularly where there is a very septic type, the range of temperature is likely to be much more irregular in time. The other symptoms must be searched for in order to obtain them. Leukocytosis in the case, the absence of the Widal reaction and diazo urine reaction, together with the physical signs, will make the diagnosis.

It is of great importance to exclude typhoid fever in doubtful cases, because it is a well-known fact that the tenderness in the right iliac fossa of simple cases of typhoid fever simulates closely the tenderness in ordinary appendicitis. It is also well known that appendicitis occasionally occurs as a part of the symptomatology of typhoid fever. The question of operation is a weighty one where typhoid fever exists.

Paratyphoid Fever.

Paratyphoid fever resembles typhoid in every respect except in the fact that a culture from the blood always shows one or another of the colon group of bacilli and the serum never gives the Widal with the Eberth bacillus, and will give a Widal reaction with some of the colon group. On these grounds the diagnosis can be made.

La Grippe (Influenza).

Especially during an epidemic of influenza, the overworked general practitioner may easily mistake cases of typhoid fever for influenza. Certain cases of influenza have a continued fever in the beginning. The onset, however, is abrupt; joint pains, headache, coryza and bronchitis are much more severe in influenza than they are in typhoid fever. In influenza the Widal reaction with typhoid bacilli is absent; agglutination reactions, however, can be obtained with the influenza bacillus. The spleen is not enlarged; there is no tenderness over the iliac fossa; the fever is less accurately regular.

Particularly the pulmonary symptoms are in the foreground of cases of influenza; they are usually slight, though present in typhoid fever.

TRICHINOSIS.

Until Brown furnished us with his conclusions, the confusion of sporadic cases of trichinosis with typhoid fever was the rule. There is the same general depression in both; continued fever and perhaps diarrhea and abdominal pains in trichinosis, but in the latter the differential blood count at once gives a clue to the diagnosis. There is a leukocytosis of from 15,000 to 30,000 white cells and a great increase in the eosinophils. This condition never occurs in typhoid fever, a case presenting leukocytosis and eosinophilia may absolutely be said not to be typhoid.

In order to make the differentiation certain, a bit of muscle may be excised under aseptic precautions. In trichinosis the embryo worm may be seen lying between muscle fibers.

OSTEOMYELITIS.

Osteomyelitis occasionally begins with fever as the main symptom, but almost without exception there is a local point of tenderness at the spot of inflammation of the bone. The presence of this and of leukocytosis, and the absence of a Widal reaction, will make the diagnosis possible. Indeed, osteomyelitis is much more often taken for rheumatism than it is for typhoid fever, because of the localized pain in or about a joint. An x-ray picture taken of a tender area should show disease of the bone in osteomyelitis.

SEPTICEMIA.

In certain cases of blood infection by various organisms, the portal of entry is often difficult to discover. Here the continued fever and the great depression and diarrhea often simulate typhoid fever (Fig. 8). In the puerperal cases the difficulty is great because, of course, puerperal individuals can develop typhoid fever.

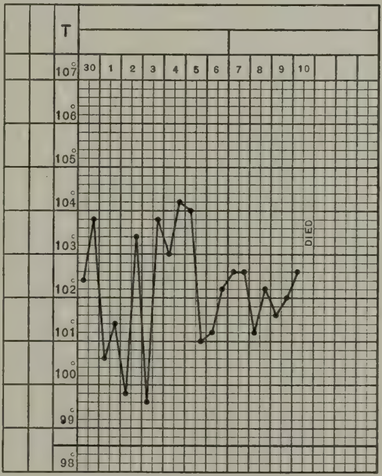

Fig. 8.—Chart of Streptococcic Bacteremia. (Personal Observation.)

The points of differentiation are: the fever due to septicemia is much more irregular than the temperature of typhoid fever; the abdominal symptoms in septicemia are, as a rule, not as characteristic as in true typhoid fever; in septicemia there is usually leukocytosis; there is no Widal reaction; a blood culture in the great majority of cases shows the organism which is causing the trouble in septicemia.

MEASLES.

The exanthemata in general, but particularly measles, may be mistaken for typhoid fever. The fever of measles begins rather abruptly. There is a fall of temperature just before the beginning of the rash which rises again with the appearance of the rash; it continues for five or six days, and then gradually declines. The case begins with coryza, bronchitis, and conjunctivitis. A rash appears about the face and neck first, and spreads gradually over the chest and extremities. Koplik's spots occur on the buccal surface a day or two before the skin rash appears. Where there is an epidemic of measles, the appearance of rather high fever, without cough or coryza, might be mistaken for typhoid, but the appearance of the rash will at once make the diagnosis. The other exanthemata can be recognized by their specific symptoms.

HODGKIN'S DISEASE.

This condition is accompanied usually by paroxysms of rather prolonged continued fever. When the case is first observed this might be mistaken for typhoid fever, but the absence of a Widal reaction, together with the presence of the characteristically enlarged glands, with a slight leukocytosis, makes the diagnosis certain. The blood picture, said by Bunting to be characteristic, is a slight leukocytosis, an increase of the transitional leukocytes and an increase of the blood platelets.

MENINGITIS.

Meningitis, particularly of the tuberculous form, is frequently mistaken for typhoid fever. Here, however, the fever is less regular; there is a very marked tache and the meningeal symptoms soon supervene. The absence of the Widal reaction in the beginning will prove a help to differentiate the symptoms. A spinal puncture will give opportunity to discover the infecting organism in the spinal fluid.

SYPHILIS.

Syphilis, occasionally in the secondary stage, is marked by a range of temperature which is not unlike that of typhoid fever; but the discovery of a rash, of sore throat, of falling of the hair, and the history or the remains of a primary sore, together with the absence of a Widal reaction, will fix the diagnosis as syphilis.

TABLE OF DIFFERENTIATION

	Temperature	*Rash*	*Condition of Bowel Movement*	*Headache*	*Delirium*	*Spleen*	*Widal Reaction*	*Blood Culture*	*Blood Count*
Typhoid Fever	Long course; gradual rise and fall	Usually scant; on abdomen; disappears on pressure	Loose	Marked in beginning	Often present	Enlarged seventh day	Present	May be positive for typhoid bacilli	Leukopenia; moderate anemia
Tuberculosis	Irregular	None	Often constipated; sometimes loose	Not marked except when fever high	Not marked	Not large	Absent	Negative except miliary tuberculosis	No increase; anemia
Endocarditis	Irregular	Petechial occasionally	Normal	Not much	When patient septic	Enlarged	Absent	Often shows organism	Leukocytes; heart murmurs
Malarial Fever	Intermittent or remittent	No rash	Normal	Intense in paroxysms	Occasionally in severe forms	Enlarged	Absent	Organisms can be grown	Organisms in blood; no change in count
Pneumonia	Abrupt rise; continues 4 to 10 days	Herpes	Normal	Often severe	Marked in severe cases	Normal	Not present	Pneumococcus	Leukocytes
Typhus Fever	Abrupt rise; abrupt fall; 10 days	Petechial	Normal	Severe	Severe	Normal	Not present	Negative	No leukocytosis
Measles	Rises 3 days, then falls and rises	Begins on face; arranged in crescents	Normal	Severe coryza; cough	Sometimes	Not enlarged	None	Negative	Normal
Septicemia	Irregular, prolonged	Sometimes petechia	Diarrhea or normal	Intense	Often present	Enlarged	Absent	Shows organism	Leukocytosis often primary
Appendicitis	Irregular	None	Usually constipated	Moderate	Moderate	None	Absent	Negative	Leukocytosis. Abdominal tenderness
Hodgkin's Disease	Continued	None	Often diarrhea	Moderate	Sometimes present	Slightly enlarged	Absent	Negative	Slight leukocytosis. Enlarged glands

ACUTE NEPHRITIS.

Acute nephritis is occasionally the initial symptom of typhoid fever. The urine is scant, contains blood, albumin and numerous tube casts. A fever is always present in this form. The Widal reaction is absent in a case of simple toxic nephritis and is present in the renal form of typhoid fever. A blood culture, positive for typhoid bacilli, will assure a diagnosis of typhoid fever with nephritis.

Conclusion

There is no single, pathologic, clinical sign upon which a diagnosis of typhoid fever can be made unless, indeed, it be a culture from the blood of the true Bacillus typhosis. Scarcely a febrile disease exists which has not at one time or another been mistaken for typhoid fever. The saddest of all errors is to mistake tuberculosis or endocarditis for typhoid fever. Valuable time is always lost, and the patient may be beyond hope of recovery, unless every possible diagnostic measure, frequently repeated, be used to help out the diagnosis. Every continued fever should be at once put to bed until a diagnosis can be made. There is no more fertile source of error than taking as absolute the absence of any one of the laboratory signs. They may be absent at one time and present at another.

2. Infection by Colon Group of Bacilli

The colon group of bacilli may cause diseases of varied character. Paratyphoid has been spoken of, and is in reality one of these colon infections. Other groups of these cases, however, are continually occurring. The symptoms may be general or they may be local. Inflammation of the gall-ducts and gall-bladder are the commonest of these colon infections. The best means of making a differential diagnosis of these colon bacillus infections is by means of a culture of the secretion, or the use of agglutinin reaction with the blood of the patient. Colon infections of the urinary tract are among the most important of all the local inflammations of this character. The parts affected are chiefly the bladder, ureters and the kidneys. The right kidney seems to be particularly affected. According to Osler, these cases are particularly frequent in children and in connection with pregnancy—and here it may occur either during or after pregnancy.

The infection is particularly obstinate, and very difficult to treat. Here, in this class of cases, it is extremely important to discover that the infecting organism is the colon bacillus, so that the treatment can be appropriately directed. The diagnosis from other inflammatory infections of these parts must depend entirely upon the culture of the colon bacillus from the discharges or from the blood.

3. Pyogenic Infection

Origin.—This condition is due to an implantation of one of the pathogenic bacteria, such as staphylococci, streptococci, pneumococci, gonococci, colon bacilli, and others. The infection takes place through a wound, a mere abrasion, a cut, a hypodermic prick, from wounds received during labor, from sutures, and so forth. Practically all of the local inflammatory disturbances, such as furuncles, carbuncles, or erysipelas, are due to infection by one of these various organisms.

So long as the inflammation remains local and there is a single boil, or one carbuncle, or a localized cellulitis, the fever and prostration which accompany the condition are not usually due to the presence of organism in the blood stream, but to soluble toxins which they have produced. When, however, there is an actual bacteriemia or an infection of the blood with any of the organisms, the case then becomes most grave, and furuncles, inflammatory foci of various character, pus formations, inflammation of the tendon sheaths and the serous membranes may occur in any portion of the body as a direct result of the blood stream infection.

The diagnosis in general infection of this character must depend upon:—

First.—The presence of a local lesion.

Second.—The presence of secondary lesions.

Third.—The recovery of the organism from the blood stream and from local lesions.

Fourth.—Leukocytosis.

Symptoms.—These last three proceedings must perforce form the basis of the diagnosis of the case. In addition to the local manifestations of the disease and to the various spreading and multiplying foci, there is fever, prostration, often diarrhea, frequently delirium, and all the symptoms of a grave bacteriemia.

Diseases to be Differentiated from Pyogenic Infection

Such a condition may be mistaken for typhoid fever, for paratyphoid fever, or if a local lesion occurs in the lungs, it may be mistaken for pneumonia—even though the infecting organism is not the pneumococcus, but because there is an actual inflammation which gives rise to the local signs of pneumonia.

There is always leukocytosis in pyogenic infections unless the patient is so overwhelmed that there is no reaction.

If the pericardium becomes infected there is, of course, an actual pericarditis, but it must be remembered that it is usually a secondary infection due to the primary condition.

The following are a few of the conditions, with their characteristic symptoms:

Typhoid Fever.

From typhoid fever the condition is separated by the fact that there can usually be discovered some focus of infection. In puerperal cases this may be impossible to definitely determine, but a continual, irregular fever following a labor case is most likely pyogenic.

The real diagnosis can be made first by the irregularity of the fever in pyogenic cases, as compared with the regularity of the fever due to typhoid infection, as shown by the chart (page 9). The Widal reaction with typhoid bacilli, is always negative in puerperal or other infections, and positive in typhoid fever. It will be positive for one of the strains of colon bacilli, if the infection is due to one of these organisms. A blood culture will determine the diagnosis if there is a true bacteriemia. There may be the same diarrhea, delirium and prostration in both cases. Infection by the colon bacillus is extremely apt to give rise to local inflammations along the tendon sheaths, in the bladder or the kidneys.

Osteomyelitis.

Osteomyelitis is constantly mistaken for some other form of infection, because frequently the local lesion cannot be made out, and the patient is treated for a general condition before the local lesion is discovered. A tender area over a bone and an x-ray, showing rarefaction, are important diagnostic points in favor of osteomyelitis.

The so-called gonorrheal and scarlatina rheumatism are really due to infection of the joints in these conditions by the particular organism involved or the result of toxemia from the organism.

Paratyphoid Fever.

Paratyphoid fever is an infection by a bacillus of the colon group, the so-called paratyphoid bacillus. As a matter of fact there may be some infection by other members of this same group, which have practically the same symptoms. The disease is almost always mistaken for typhoid fever, because of the absence of all local lesions in any portion of the body, which would account for the fever; and the absence of Widal reaction with typhoid bacillus show it to be something else than true typhoid fever.

The symptoms are fever with a slightly less shorter incubation period, a fever lasting not so long as true typhoid fever, fewer systemic symptoms—that is, less delirium, less tendency to coma, less diarrhea, and less abdominal distention. The fever runs a regular course from two to three weeks exactly like typhoid fever, gradually dropping to normal in a few days. There is no leukocytosis; there is absolutely no Widal reaction with the typhoid bacillus after repeated attempts to obtain it.

The fever can be mistaken for practically only typhoid fever, and after one is sure, by the absence of leukocytosis and the absence of local signs in other portions of the body that there is no local focus of disease, the diagnosis can be made certain by the absence of Widal reaction with the Eberth Bacillus and the presence of a clumping reaction with one of the group of colon bacilli, also by the recovery of the organisms by blood cultures either from the blood or cultures from the spleen, urine or feces.

4. Erysipelas

Characteristic Features.—Erysipelas is an infectious disease characterized by a local inflammation of the skin, high fever, exhaustion, and sometimes delirium. The infecting organism, a streptococcus. The Streptococcus erysipelatis, which is doubtless the same organism as Streptococcus pyogenes, gains entrance through an abrasion or a wound in the surface of the skin. There is leukocytosis.

The type of erysipelas which affects principally the face—facial erysipelas—is so characteristic a disease, though probably due to the same organism which causes erysipelas in any other portion of the body, that it deserves especial notice.

Course of the Disease.—It is frequently ushered in by a chill, soon to be followed by characteristic symptoms. Occasionally the first symptom is a spot—red, tender, raised above the surface of surrounding skin, hot to the touch, and often situated on the bridge of the nose. The edges of this inflamed area are remarkably abrupt, being raised above sound skin. The swelling rapidly extends (in twenty-four hours the entire face may be swollen out of recognition), the eyes close, the lids become edematous, the lips swell, and the whole face becomes red and glistening. Within forty-eight hours the entire red area is covered with blebs containing serum, the blebs varying in size from that of a pin's head to the size of a hen's egg. The glands draining the infected area are enlarged.

Immmediately after the chill the temperature rises to 101° to 104°, and even up to 106° F., continuing for five or six days, when it gradually but rather quickly subsides to normal.

The patient is much distressed, and often delirious. The chief distress is the terrible burning and itching of the face. Sometimes, indeed usually, the scalp becomes involved, and may be the seat of deep subcutaneous abscesses. The mucous membranes are rarely affected. In other portions of the body the eruption usually begins about a spot of abrasion. In facial erysipelas there is probably also a small local abrasion or other wound which acts as the focus of infection, but it has rarely been the lot of the writer to discover such a spot.

The urine often contains albumin.

Diseases to be Differentiated from Erysipelas

It may be mistaken for the following conditions:

Cellulitis
Redness and tenderness overlying a deep focus of pus
Simple erythema
Erythema nodosum
Urticaria
Eczema.

Cellulitis.

In cellulitis there is infiltration of the substance of the tissues underlying the skin. The skin is glazed and red and does not have a sharp demarcation between the inflamed area and skin. Fever, leukocytosis, and local tenderness over the area, are very marked. Underlying the painful area fluctuation is often found, and there is not apt to be the formation of blebs in the skin as there is in erysipelas.

The condition is more apt to be mistaken for erysipelas in other portions of the body than the face.

The Redness and Tenderness Over a Focus of Pus.

The redness and tenderness over a focus of pus is usually circumscribed. Fluctuation can be felt beneath the inflamed area. Blebs are not present and there is no sharp demarcation between the sound skin and the inflamed area.

Simple Erythema.

Simple erythema does not infiltrate the skin; is never covered with blebs; the constitutional symptoms are very slight.

Erythema nodosum.

Erythema nodosum occurs usually upon the legs. There are actual nodules which can be felt beneath the skin, which are extremely painful. The color is usually a dark red or bluish-red, instead of the bright and inflamed area that occurs upon the erysipelatous skin. The patient may have fever, leukocytosis and joint pains. There is leukocytosis.

URTICARIA.

There are practically always gastro-intestinal symptoms. The rash appears quickly and disappears within a few hours. Itching is very great while it lasts, but disappears in a few hours. There are no blebs.

ECZEMA.

Eczema, especially when there is an acute exacerbation of a chronic condition, might be mistaken for erysipelas, but there is this very marked difference in the two conditions: eczema is purely a local condition without any constitutional involvement, unless there be some deep-seated suppuration underlying an eczematous patch; this sometimes occurs when the patient infects the eczematous area by scratches. Eczema is itching. The blebs, when they occur, are merely small vesicles, and not bullae as in erysipelas. There is leukocytosis in erysipelas, and *not* in eczema.

TABLE OF DIFFERENTIATION

	Skin Eruption	*Blood Examination*	*General Symptoms*
ERYSIPELAS	Bright red, infiltrated Demarcation Bullae	Leukocytosis. Streptococci can be found in blood	Fever. Prostration Delirium
REDNESS OVER PUS, ETC.	No demarcation Fluctuation. Glazed No bullae	Leukocytosis	Fever and delirium ceases with excavation of pus
SIMPLE ERYTHEMA	No demarcation No bullae	No leukocytosis	No fever; patient feels well
ERYTHEMA NODOSUM	Nodular, painful No bullae on legs	Leukocytosis	Fever Tenderness over spot
URTICARIA	White or red; evanescent	No leukocytosis	Gastro-intestinal symptoms
ECZEMA	Itching No demarcation	No leukocytosis	No general symptoms

5. Diphtheria

Origin.—Diphtheria is an acute transmissible disease due to infection with the Bacillus diphtheriæ (Klebs-Loeffler Bacillus).

The site of election of inflammation of the bacillus is the throat, though any mucous membrane may be the original seat of the disease, or the portal of entry may be through an abraded spot on the skin.

In the beginning the disease is purely a local one. The degree of the constitutional disturbance and the rapidity of its appearance depend upon the amount of toxin produced and absorbed.

The disease may be transmitted directly from one individual to another, through the medium of discharges from the affected part of the patient coming in direct contact with the well—as by coughing, sneezing, or kissing, or through the medium of the hands; secondarily by any article which has come in direct contact with the infective material from the throat and nose.

Bacillus of Diphtheria.—The bacillus of diphtheria (Plate 1) is a short rod, often clubbed at the end, and staining irregularly. In cultures from the throat it is apt to be arranged in parallel bunches. Its dimension is 5 to 8 micromillimeters in length, and 2 to 3 in width. It grows best on Loeffler's blood serum. It is stained well by Loeffler's blue, and a good differential stain is Neisser's. In the two cultures figured on plate, taken from original cases, the mode of growth in the tube, and the appearance on staining can be seen. The blood shows a secondary anemia, and in severe cases a leukocytosis.

General Symptoms.—The symptoms are a feeling of fullness in the throat, malaise, headache, moderate degree of fever, sometimes vomiting and aching of the limbs.

The feeling of fullness in the throat may be an actual soreness with difficulty in swallowing, which appears rapidly. The fever may be high and vomiting severe; there may be convulsions, with early unconsciousness, especially in children.

The glands at the angle of the jaw rapidly swell, and may form great tumors which are more or less painful. Very early there is a bricklike redness over the whole fauces.

Exudate.—Soon, and usually when the case is first seen by the attending physician, there is an exudate, usually attacking the tonsils first, but rapidly spreading to the soft palate, to the pharynx and to the roof of the mouth. This exudate is a true membrane, the edges fading into the inflamed mucous membrane and is tightly adherent to the underlying mucous membrane. Any attempt at removal of the membrane leaves a raw and bleeding surface beneath.

A SMEAR made from this exudate and examined, shows the presence of the characteristic bacillus of diphtheria. According to McCollom the smear is best made from the edges of the exudate.

A CULTURE made from the throat (and in the author's experience this is the best plan) will show within eight or ten hours, certainly within eighteen hours, a practically pure culture of diphtheria bacilli.

Membrane.—The membrane is first grayish white, or it may be even pearly white in appearance, turning grayish, and even greenish or black. It may be easily broken up, or a thick, tenacious, fibrous membrane may form. The following description is taken from McCollom:

"The local lesions show in all situations the same pathological process. Many degenerative changes in the epithelial cells and in the underlying tissues, combined with an abundant fibrous exudation from the blood vessels—as a result a membrane is formed in the epithelial surface."

Its removal from the underlying tissue shows a loss of substance and

PLATE I

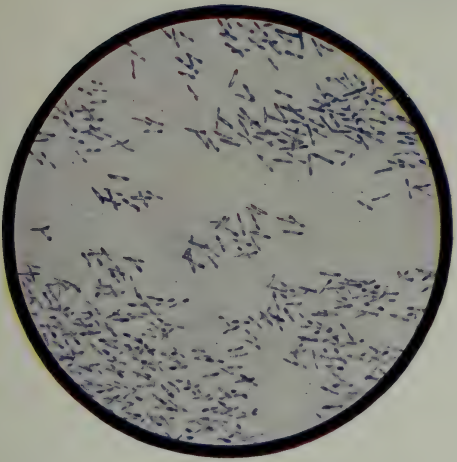

Fig. 1.

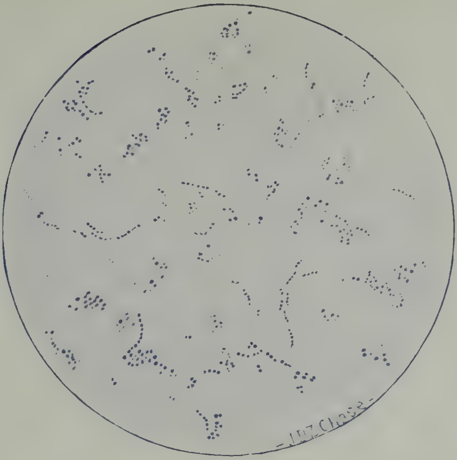

Fig. 2.

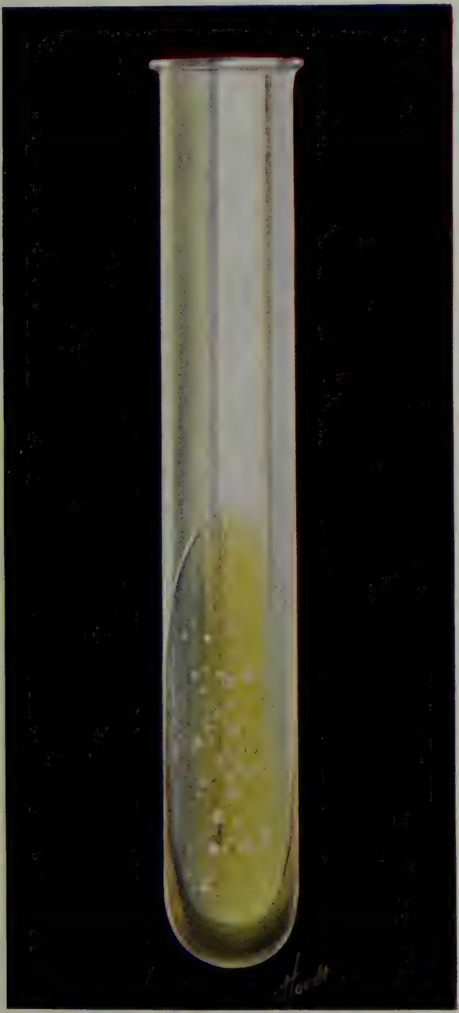

Fig. 3.

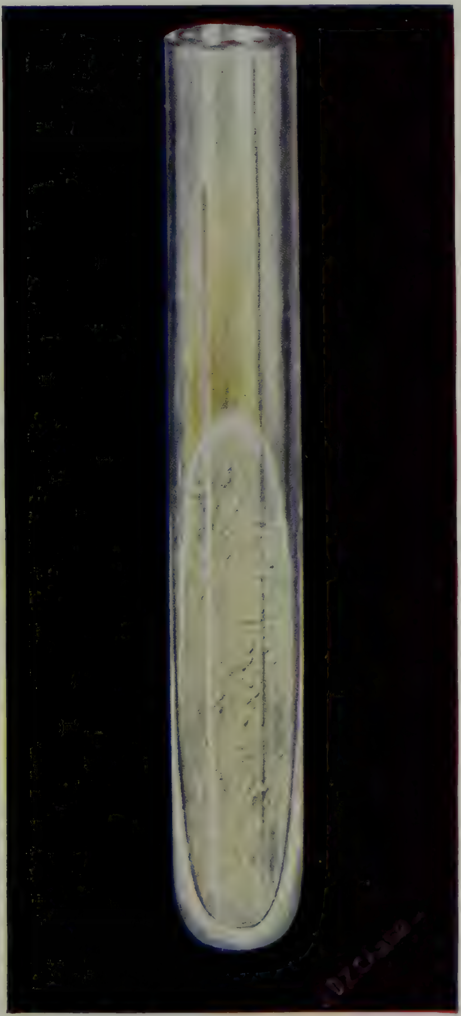

Fig. 4.

Fig. 1.—Diphtheria Bacilli from Culture.
Fig. 2.—Culture from Throat of Follicular Tonsillitis. (Personal Observation.)
Fig. 3.—Diphtheria Bacilli Growing on Agar-agar.
Fig. 4.—Culture Tube from Follicular Tonsillitis. (Personal Observation.)

often there is bleeding of the diseased surface. The membrane instead of spreading may remain absolutely localized to the tonsils, even in the follicles of the tonsils, and yet be true diphtheria, as was pointed out long since by Jacobi.

If the case is not treated, exhaustion is extremely rapid, and frequently extreme. As the case progresses, untreated, a foul odor appears on the breath. The patient lies with his mouth open; there is stertorous breathing; bloody discharge appears at the nostrils, which excoriates the skin of the upper lip. The patient becomes semiconscious, and can scarcely be aroused for food or drink. The heart is always rapid, frequently running to 120 beats to the minute.

Before the use of antitoxin, this picture was frequent enough. Now it is happily rare, when cases are treated early, and with full doses of antitoxin.

Paralysis.—If the case is untreated with antitoxin, and occasionally even though antitoxin be used, there will be a late paralysis. This usually affects the soft palate first, so that there is inability to swallow properly, all liquids being regurgitated through the nose. This paralysis, which is due to a peripheral neuritis, occurs very rapidly, and may affect practically every muscle in the body. The paralysis may be so slight, however, that it is indicated only by a disappearance of the reflexes.

Vision may be affected by paralysis of the ciliary muscle, or by paralysis of the ocular muscles, the latter giving rise to diplopia.

Special Symptoms.—The Heart.—The involvement of the heart is due either to a toxic myocarditis, or to a neuritis. Sudden death frequently supervenes upon a severe exertion. No case of diphtheria, be it ever so light, can be looked upon as free from this danger.

The Kidneys.

The kidneys are always more or less involved. Usually there is albuminuria. Occasionally there is a true nephritis, which may become a genuine complication.

The Lungs.

The lungs may become the seat of a bronchopneumonia, and this complication accounts for many deaths in the course of an epidemic. The consolidation, according to McCollom, may be due to a growth of the Bacillus diphtheriæ. The diagnosis between this true diphtheria of the lungs and a bronchopneumonia can only be made by finding diphtheria bacilli in the sputum from the lung.

Bacteriological Examination.—A culture should be made from every suspicious sore throat, in just such a routine manner as a urine examination should be made in every case treated for any disease. There is no single symptom; no peculiar appearance of an exudate due to diphtheria, which will allow a correct diagnosis to be made every time, unless a culture be taken, and *occasionally more than one culture must be made.*

If cultures were made in a routine manner, fewer cases of diphtheria would be wrongly diagnosed; many epidemics would be prevented, for notoriously, cases of tonsillitis, supposed to be rheumatic, etc., are fertile sources of beginning epidemics of diphtheria. Many boards of health now give ample opportunity for every physician to have every case examined bacteriologically. *Every physician should be able to examine his own specimens.*

On November 16, 1901, in the Journal of American Medical Association, the author called attention to the necessity for this. Increasing experience has shown its necessity still more. The materials necessary are a knowledge of the microscope and how to make cultures.

Outfit.—A culture medium, preferably blood serum, can be purchased from the manufacturers of biological products or from any manufacturing chemist. Loeffler's methylen blue solution and a small oven with a thermostat are essential. The entire outfit, exclusive of the microscope, can be obtained for ten dollars. Instead of a thermostat an ordinary thermos bottle may be used for keeping the culture at the proper temperature.

Culture tubes should be carried by the physician in his implement bag. A diagnosis can be made in twelve hours. Occasionally there occurs in the throat of normal individuals, bacilli which in cultural and staining qualities exactly resemble diphtheria bacilli, but differ from them in virulence.

Authorities are agreed that it is a mistake to call these organisms pseudodiphtheria bacilli, as they are probably only a strain of the true bacillus of low virulence.

Cultures of the throat are better than smears, and in eight to ten hours the diphtheria bacilli can frequently be found.

Laryngeal Diphtheria

The larynx is frequently affected, sometimes giving rise to the so-called membranous croup (a name now to be discarded). In this condition there is hoarseness, often amounting to aphonia—a peculiar indisposition on the part of the individual to attempt to speak, usually inability to sound a high note—the cry, therefore, being smothered and dull.

If the laryngeal stenosis becomes extreme, there is great difficulty in breathing, long drawn difficult inspiration and expiration, with depression of the epigastrium and margin of the ribs, cyanosis and, if the condition is not relieved, death from suffocation.

Laryngeal diphtheria must be differentiated from other conditions causing stenosis of the larynx. In acute cases this is always a difficult task unless the stenosis is a symptom of some chronic condition, or the laryngeal symptoms accompany a frank faucial diphtheria.

Diseases to be Differentiated from Laryngeal Diphtheria

It can be confounded with:

Catarrhal or spasmodic croup
Laryngismus stridulus
Edema of the larynx
Exudative laryngitis—not due to diphtheria
Postdiphtheritic paralysis of the vocal cords
Stenosis the result of pressure from a tumor, or due to nerve paralysis by tumor
Postpharyngeal abscess, foreign bodies.

The following are some of the conditions with their special symptoms:

ORDINARY SPASMODIC CROUP.

Ordinary spasmodic croup is almost without exception sudden in onset, and occurs usually in the middle of the night. Frequently there have been symptoms of a faucial catarrh during the day, preceding the attack. In diphtheritic croup there has frequently been observed some exudate on the tonsils or fauces antedating the attack. The diphtheritic attack is rather a gradual increasing hoarseness and stenosis than a sudden onset of difficult breathing. The individual with spasmodic croup is hoarse but is quite able to cry aloud and often able to make a high note. The child with diphtheritic laryngitis has a smothered voice, though the cough may be high pitched. If there is an exudate present in the throat, the chances are the case is diphtheritic, and the case should be treated as such. Finally, cultures may be made, but are very apt to be negative, because the swab frequently does not come in contact with the exudate on the vocal cords.

LARYNGISMUS STRIDULUS.

Laryngismus stridulus is characterized by a tonic spasm followed by crowing inspirations; it is chronic in character and is often an effect of rickets or tetany. There is neither catarrhal condition of the fauces nor exudate in the throat.

EDEMA OF THE LARYNX.

Edema of the larynx comes from many causes, whether due to a perichondritis, to a nephritis, or a nearby abscess; it is usually more chronic in onset than diphtheritic croup. If due to perichondritis or a retropharyngeal abscess, there has been tenderness locally or pain, and often hoarseness for some time. There is no local tenderness or pain in laryngeal diphtheria. If the edema is due to nephritis, there are the urinary symptoms of nephritis.

Exudative Laryngitis.

Exudative laryngitis, due to other organisms than Klebs-Löffler bacillus, while rare, does occasionally occur. It cannot be diagnosed without cultural methods. When acute laryngitis occurs in measles, it may be mistaken for diphtheria, but here the ordinary symptoms of measles are present.

In certain mediastinal *tumors* there is pressure on the trachea which causes a stridor not unlike that in diphtheritic laryngitis, but there is no hoarseness and the condition is of long duration.

Paralysis of the Recurrent Laryngeal Nerve.

This may be mistaken for croup, but the condition is always the result of some pressure on the trunk of the nerve or of the result of an acute infection, where there is either inflammation of the center, or there is a neuritis. The author has seen a postdiphtheritic paralysis, with paralysis of the cord, bring on attacks not unlike diphtheritic croup.

Retropharyngeal Abscess.

This condition comes on slowly; there is no exudate, and the cough is not croupy. Examination of the pharynx with the finger will show a fluctuating mass in the pharyngeal wall.

Foreign Bodies.

When a body is inspired and lodges in the larynx or bronchus, the symptoms follow immediately upon the accident, and are not gradual in their onset. Hard metallic bodies can be made out by the x-ray.

Thymic asthma and enlarged bronchial glands sometimes give rise to stridor not unlike that of croup, but the voice is clear, and the mass under the sternum can be made out by percussion or by x-ray.

In all these cases examination of the larynx is of the greatest value, but the usual physician is rarely able to make a satisfactory examination of the larynx with a mirror, in a dyspneic and struggling individual.

Nasal Diphtheria

A peculiar membranous rhinitis occurs in which both anterior nares may be literally filled with a thick, leathery membrane difficult to remove.

Ravenal cites 77 cases, in 41 of which bacteriological examinations were made. Of these 41, 33 showed the presence of diphtheria bacilli. This form may give rise to cases of diphtheria in persons in contact with the disease. It is rarely fatal.

There is another form of nasal diphtheria, however, in which the implantation takes place in the posterior nares. Here there is a bloody discharge from the nares. The constitutional symptoms are severe. The exudate frequently does not make its appearance where it can be seen until the patient is extremely ill. This is often a very fatal form of diphtheria, death being due to the extreme toxemia.

The eyes are occasionally affected. The conjunctiva is attacked. The severe cases may result in entire destruction of the sight, and even of the eye itself.

The genitals are occasionally involved. The author has seen a case where the vulva and vagina were the seat of an extensive exudate—the result of secondary infection.

The middle ear may become affected, giving rise to all the complications of suppurative middle ear disease.

Hemorrhage in severe cases may occur from the mucous membranes or appear upon the skin. The latter is of the most serious import, being the result of hemolysis caused by the diphtheria toxin.

Diseases to be Differentiated from Diphtheria

Diphtheria must be differentiated from:

Follicular tonsillitis
Various anginas with exudate—usually streptococcic
Vincent's angina
Scarlet fever
Measles
Syphilis
Parasitic stomatitis
Peritonsillar abscess
Serum sickness.

In most cases the combination of the typical local sign of membrane, coupled with the physical signs will enable one to make an acccurate diagnosis. However, the final test must come through the routine use of throat culture, as stated above.

Follicular Tonsillitis.

This is unquestionably an infectious disease, ushered in by aching limbs, a distinct chilly feeling, frequently with high fever. The exudate, unlike that of the usual case of diphtheria, is confined to the tonsils, often to the follicles of the tonsils, though a thick pultaceous mass may cover the whole tonsil. The exudate is easily detached, and does not leave a bleeding surface behind it. A culture usually shows a staphylococcus or streptococcus in almost pure culture. As has been stated, this culture is the only positive diagnostic sign. Frequently the solitary follicles on the back of the pharynx are affected by the same process.

Nondiphtheritic Exudative Angina.

The title is here adopted in preference to pseudodiphtheria. The exudate is frequently a true membrane, and may leave behind a bleeding surface. The condition may be primary, and is frequently of great severity and is then due to primary implantation of a streptococcus or staphylococcus; or it may be present in the course of measles, scarlet fever, whooping-cough, etc. Many of the supposed mixed cases of scarlet fever and diphtheria are due to a membranous pharyngitis, caused by a streptococcus or staphylococcus. When the condition is due to a streptococcus there are frequently extremely severe constitutional symptoms.

Vincent's Angina.

This is characterized by painful swelling of the fauces, and ulceration of the tonsils, covered by a foul-smelling exudate. In appearance it is not unlike diphtheria of rather more chronic course. Examination of a smear shows the presence of a spirochete, and a fusiform bacillus. The exudate may appear on the oral mucous membrane at other points than the tonsils. The ulceration has a peculiar "punched out" appearance, differing in this respect from diphtheria.

Scarlet Fever.

This begins, so far as symptoms are concerned, in the same manner as diphtheria, except that as a rule all the symptoms are more severe, even when the case is a light one. The exudate on the throat when it is present is usually in streaks of grayish white material. Within twenty-four hours there is the characteristic scarlet rash, beginning first on the chest and rapidly spreading over the trunk and limbs. Cultures from the throat are negative for diphtheria bacilli.

Measles.

Measles is less likely to be mistaken for diphtheria, unless the case be complicated with an exudate early in the course. There is a red throat spotted over with a typical rash of measles. On the buccal mucous membrane are the characteristic Koplik spots, which appear on the first day before the skin rash. The culture is negative for diphtheria bacilli. There are symptoms of bronchitis and coryza.

SYPHILIS.

Syphilitic sore throat may also be mistaken for diphtheria. In this there is enlargement of the posterior, cervical, inguinal and epitrochlear lymphatic glands, with the usual secondary rash, and always the absence of the bacilli of diphtheria. The Wassermann test is positive.

APHTHOUS STOMATITIS.

This condition is characterized by punched out ulcers occurring in the mucous membrane of the cheeks and lips. The ulcers are painful. There is but little general depression.

THRUSH.

This is occasionally taken for diphtheria, especially when the exudate is extreme in amount; then the new growth forms a veritable membrane, which usually covers tonsils and the soft palate. The least touch, however, serves to remove great patches, and the microscope shows mycelium and spores of the characteristic growth. None of these conditions are followed by postdiphtheritic paralysis. The use of antitoxin is without effect in any but diphtheritic conditions.

PERITONSILLAR ABSCESS.

Peritonsillar abscess is simulated by acute local edema not uncommon in diphtheria. In peritonsillar abscess there is great difficulty in opening the mouth. The condition comes on more slowly. A puncture will show the presence of pus and above all the culture does not show diphtheria bacilli.

The exudate which usually appears after the removal of the tonsils may be mistaken for diphtheria, but the fact of a previous operation and the absence of signs of toxemia together with the absence of diphtheria bacilli, make the diagnosis.

SERUM SICKNESS.

Since the universal use of antitoxin, there has arisen a series of symptoms—the result of the employment of that specific remedy—which has now received the above name.

Firstly, there are local symptoms. In certain individuals the whole back or other area where the injection has been made is exquisitely tender, slightly swollen and red. The patient lies curled on his side, and the slightest attempt at movement causes him to resist and cry out. It is extremely rare that suppuration occurs at the seat of injection, and when it does occur it is unquestionably due to secondary infection and has no relation to the specific effect of the foreign serum.

Erythema resembling urticaria, scarlet fever or measles, occurs sometimes as does a general painful condition of all the joints, without swelling or redness. Sudden death has supervened in a small number of cases, but the unfortunate accident would seem to have nothing to do with the antitoxin itself, but is only the unfortunate result that occasionally follows the injection of any foreign proteid—the so-called **anaphylactic reaction**.

This serum sickness should not be mistaken for a complication of diphtheria, but should be looked upon as the direct anaphylactic result of the foreign proteid, in the shape of horse serum.

6. Croupous Pneumonia

Characteristic Features.—Croupous pneumonia, "Captain of the Men of Death," is an infectious disease characterized by a consolidation of one or more lobes of the lung. A pneumococcemia occasionally occurs, however, in which the lung lesion may be delayed or even wanting.

The Organism.—The cause of the infection is an organism generally recognized under the name "Pneumococcus or Diplococcus Pneumoniæ." This organism has been uniformly found in the solidified lung of pneumonia, and in many instances in the blood of the living individual suffering from pneumonia. It is uniformly present in the inflammatory complications of pneumonia. "The organism is commonly found as a diplococcus, though here and there short chains of four to six individuals may be detected. The individual cells are more or less oval, or more strictly speaking, lancet shaped, for at one end they are commonly pointed. When joined in chains the junction is always at the broad ends of the ovals, *never* at the pointed extremities. When in chains, only the terminal cells are pointed, and then at their distal extremities" (Abbott).

General Symptoms.—The onset is usually sudden and ushered in by a chill; occasionally the onset is more gradual. The individual may feel out of sorts, and suffer from a bronchial catarrh for several hours or days before the initial chill, or the chill may be absent and the patient gradually develop fever, dyspnea and pain as the first symptoms of serious trouble. Coincident with the chill there is fever. The temperature is raised during the chill, and this initial temperature rises within an hour or two to the height of 103° or 104° F. In children there is frequent vomiting, and perhaps delirium and convulsions. Frequently there is a sharp stabbing pain over the area of the lung affected—most commonly at the base. In the majority of cases there is a short, sharp, unproductive cough present in the beginning. The cough and pain soon increase and

TABLE OF DIFFERENTIATION

	Onset	*Appearance of Throat*	*Throat Culture*	*Symptoms*	*Result*	*Effect of Antitoxin*	
DIPHTHERIA	Sudden	First brick red; then a membrane on tonsil and fauces	Positive for Klebs-Löffler	Slight fever; sore throat; muffled voice	May be fatal	If antitoxin is given early, cases cured	
FOLLICULAR TONSILLITIS	Sudden	In follicles of tonsils, or covers whole tonsil	Negative for Klebs-Löffler	Higher fever; prostration	Not fatal, except by sequelae	No result	Peritonsillar abscess is apt to occur
THRUSH—PARASITIC STOMATITIS	Gradual	White; usually in patches	Negative for Klebs-Löffler bacilli Mycelium and spores	Digestive and general weakness	Not fatal	No result	
SYPHILIS	Initial lesion	Red; some exudate; no membrane	Negative for Klebs-Löffler	Rash. Alopecia Primary sore	Recovery if treated	No effect	Shows Wassermann reaction
NON DIPHTHERITIC EXUDATIVE ANGINAS	Sudden	Membrane in any portion	Negative for Klebs-Löffler	Often severe	Usually recover	No effect	
VINCENT'S ANGINA	Rather gradual	Red; punched out; some exudate	Spirillum and fusiform bacilli	Often severe	Curable	No effect	
EXANTHEMATA	Sudden	Red; swollen; measles. Koplik spots	Negative for Klebs-Löffler	Severe. Rash, coryza, etc.	Often fatal	None	

	Onset	*Voice*	*Progress*	*Effect of Emetics*	*Effect of Antitoxin*	*Breathing*	*Culture*
DIPHTHERITIC LARYNGITIS	Within 24 to 48 hours	Smothered	Toward death unless treated	Temporary improvement	Cure	Difficult inspiration and expiration	Diphtheria bacilli
SPASMODIC CROUP	Very sudden	High-pitched; hoarse	Toward recovery	Immediate cure	No effect	Noisy	No diphtheria bacilli
LARYNGISMUS STRIDULUS	Gradual	Little change	Recovery by general treatment	No effect	No effect	Noisy; comes in spells	No diphtheria bacilli
EDEMA OF LARYNX	Sometimes sudden	Smothered	Toward death	Little effect	No effect	Smothered and difficult	No diphtheria bacilli
STREPTOCOCCIC OR OTHER LARYNGITIS	Resembles diphtheria	Resembles diphtheria	Toward death	Temporary improvement	No effect	Resembles diphtheria	No diphtheria bacilli
PARALYSIS OF CORDS	Usually gradual	Often whispering	Cured only by removal of cause	None	None	Usually inspiration difficult	No diphtheria bacilli

the patient suffers from dyspnea, but chiefly from the excessive pain suffered. Soon the face becomes flushed, the patient is inclined to lie on his back, and the breathing becomes short and quick.

If the case is a sthenic one and no complications occur, the temperature remains high, ranging from 103° to 105° F. for a period of from four to eight or nine days. It then suddenly falls to normal within twenty-four hours (the crisis) or gradually reaches normal within three or four days (the lysis).

Special Symptoms.—The Chill.—This symptom is extremely common. In 12,402 cases (Musser and Norris), 58.12 per cent of the patients had a chill; 30.12 per cent suffered from chilliness. During the chill the patient suffers extremely, the teeth chatter; the limbs shake; the patient feels as though he were exposed to extreme cold. The lips and finger tips are often cyanosed.

Temperature.—The temperature usually rises to the fastigium within a few hours of the onset of the disease and remains high until convalescence occurs, either beginning by crisis, a sudden drop from high fever to normal within a few hours (Fig. 9), or more slowly, reaching the normal mark within two or three days (Fig. 10). Occasionally—and this is especially the fact in childhood—the beginning of the temperature may be characterized by marked remissions or intermissions, but after three or four days the temperature remains uniformly high. Sometimes there is precritical rise of temperature. When this occurs, the other symptoms of the

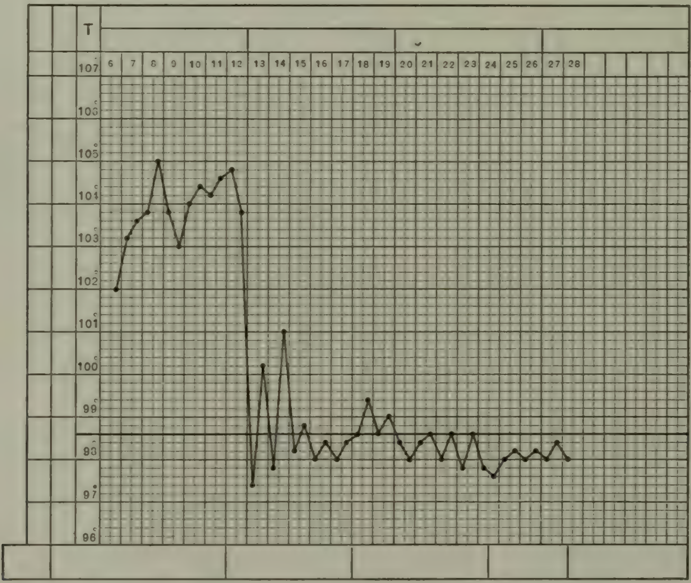

Fig. 9.—Chart Showing Crisis of Pneumonia. (Personal Observation.)

disease increase in severity, and the patient may seem in urgent peril of his life.

There is occasionally also, a pseudocrisis. Here the temperature falls as in a true crisis, but the other symptoms are as a rule not commensurately improved. After the low point has been reached there is a rapid rise to the normal height of the fever. After crisis or the fall to normal by lysis, the temperature as a rule remains normal. If there is a subsequent rise, either some complication is occurring or the very rare relapse occurs.

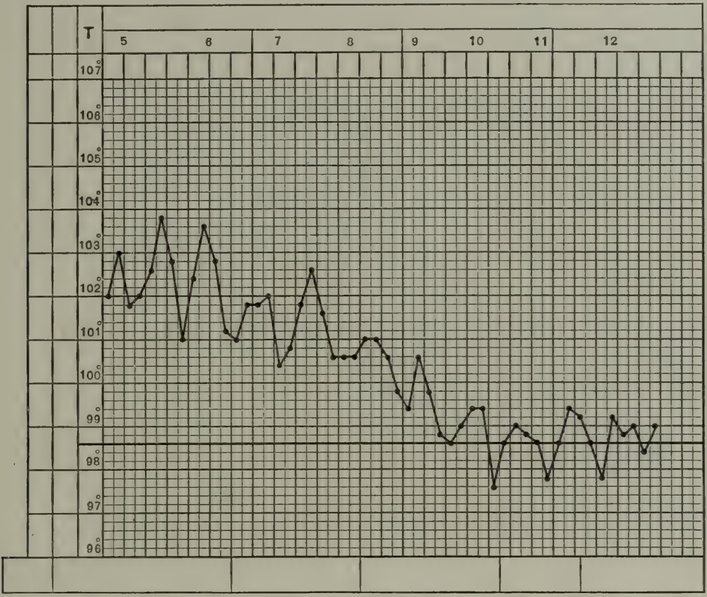

Fig. 10.—Chart Showing Lysis in Pneumonia. (Personal Observation.)

Pain.—Pain occurs in the great majority of sthenic cases; it is either extremely sharp and cutting and causes the patient to moan with each respiratory movement, or it is dull in character. In children and in the aged, pain may be entirely absent. The seat of the pain, as a rule, is over the part of the affected lung but it may be reflected, notably to the abdomen. Many instances are on record, where the first symptom complained of is pain in the abdomen, either over the appendix or gall-bladder, causing the condition to become confused with appendicitis or cholecystitis. Occasionally the abdominal pain is diffuse and the abdominal distention is so great that the condition is taken to be an acute inflammation of the peritoneum.

Bennecke (Med. Klinik, 1909, No. 7, p. 244) reports 21 cases with abdominal symptoms. In 9 of these cases the right upper lobe of the lung was involved, and in 7 the right lower lobe was the seat of the disease.

Cough and Expectoration.—At first the cough is short, sharp and unproductive; later there is more sputum, which at first is simply mucus, becoming purulent and colored with blood—the *rusty* sputum. If the blood is profuse the sputum becomes dark and of the so-called "prune juice" type. Sometimes this "rusty or prune juice" sputum occurs in the first forty-eight hours of the disease.

During convalescence the cough and sputum diminish. The sputum is markedly slight in children. Otten observed sputum in but 15 out of 250 cases reported in children. Occasionally, and this is particularly frequent in children, there is an entire absence of cough.

Herpes.—Herpes occurs in a large percentage of cases, usually about the lips—though the author has notes of several cases where the eyelids and forehead were affected. It may occur over the distribution of any of the nerves, the fifth, however, being the most common.

Laryngitis.—Laryngitis has been a severe and annoying symptom in a certain number of cases. If this symptom alone claims attention of the physician, error is certain to arise.

Diarrhea and Dysentery.—Diarrhea and dysentery have been present both as initial symptoms and as complications.

Vomiting.—Vomiting is a frequent symptom in the onset, especially in children. It usually disappears early, but may last through almost the entire course of the disease.

Respiration.—Respiration is usually hurried. The respiration pulse ratio usually in health—one to four—is often one to three, one to two, and in extreme instances (one notably the author's) the respiration was between 75 and 80 per minute for several hours in a case which recovered. Frequently there is a moan with expiration, the so-called expiratory grunt, so common in children.

Pulse and Blood Pressure.—The heart's action in sthenic cases is rapid and often strong under such circumstances. The pulse is rapid and full. If the pulse becomes rapid and weak in the course of an attack, the case is failing. A weak, irregular, and rapid pulse is common in the aged and weak, and is of bad omen.

The blood pressure has no constant relation to the course of the disease. However, if there is a decided and rapid fall, and this low pressure continues, there is great danger of collapse; on the other hand, as pointed out by Janeway, there may be a decided rise when the patient is extremely ill and cyanotic.

The Heart.—The action of the heart is always quickened. Usually the first sound is strong and booming, and the second sound is accentuated. Disappearance of the accentuation of the second sound may be a very unfavorable sign. Pericarditis has occurred in certain cases. Murmurs due to pure functional disturbance are common. Endocarditis with its consequent murmurs is rare. Dilatation may occur gradually or suddenly, and is always of bad import.

Urine.—In the vast majority of cases the urine contains albumin. It frequently contains tube casts. The chlorids are said to be constantly diminished. Occasionally there is a true nephritis, characterized by suppression of urine and a large amount of albumin and tube casts. It occasionally dominates the case. The mere appearance of albumin and a few casts is of no importance.

The Blood.—Until after the crisis there is little change in the red cells or hemoglobin. Afterward there is a slight anemia. Almost without exception there is an increase of leukocytes in the beginning of the disease, the degree of leukocytosis being usually determined by the degree of infection and the resistance of the individual. Very mild cases may have a very slight increase of the leukocytes, certain asthenic grave cases have so little reaction, that no leukocytosis is present. Cabot says that a lack of leukocytosis means one of the two above conditions. Etienne and Perrin (J. de Physiol. et de Path. Gen., Paris, 1909, XI, pp. 58-72) reporting cases in the aged, claim an average of 15,000 to 20,000 leukocytes to the cm., and found no relation between the gravity of the disease and the number of leukocytes. In 39 cases at St. Timothy's Hospital, Roxborough, in which the leukocytes were counted, the fatal cases had an average of 15,000, with one exception, here there were 8,000. Eosinophils are usually decreased; their increase is said to be favorable. Their return to the peripheral blood after they have disappeared, is a good omen.

The leukocytosis generally increases immediately with the chill, and disappears with the crisis or lysis. It does not diminish with a pseudocrisis. The return of leukocytosis after a crisis, or its continuance, means almost always an inflammatory complication. Leukocytosis is of great diagnostic value in cases where physical signs are scant, but where chill, fever, pain and cough indicate some acute pulmonary condition. It is absent in typhoid fevers, malarial fever and influenza.

The pneumococci may be found in the blood stream, in children less commonly than in adults. They disappear from the blood stream just before the crisis. Olten found pneumococcemia in 9 out of 76 cases examined in children. Prochaska (Deutsch. Arch. f. klin. Med., 1901, vol. LXXX, p. 550) found it in almost every case. Probably individual technic has much to do with success in finding these organisms. Such has been my experience with men who have searched for them.

The red cells are rarely much diminished except after the crisis or some severe complication. The appearance of normoblasts in the peripheral blood has been noted by Cabot and others.

Nervous System.—In children, a convulsion or reflex vomiting with delirium is common in the beginning of the disease. Throughout the disease the child is either extremely restless, tossing about and threatening his life by his activity, or on the contrary he may be so overwhelmed with poison that he is in a constant semiconscious condition. Frequently meningeal symptoms supervene, which are simply the result of the effect of the poison on the brain; these are the so-called cases of *meningismus of Kirschtheim.* True meningitis may supervene. This will be spoken of later.

Types of Croupous Pneumonia.

STHENIC TYPE.—In this condition the patient is suddenly stricken; all the symptoms and signs are quickly developed—the fever is high, the pulse is strong, full and rapid, leukocytosis is high, and the involvement usually great.

ASTHENIC TYPE.—The patient is partially collapsed; the pulse is rapid and feeble; the temperature is not high; the heart has an early tendency to dilate; cyanosis occurs and the lung involvement may be great or small.

Various Forms of Pneumonia.

(1) SENILE PNEUMONIA.—Senile pneumonia is often insidious in its onset. The patient may be extremely ill with a large amount of lung involvement, before symptoms attract the attention of the patient or his friends. On the other hand, a series of cases of pneumonia in the aged studied by Etienne and Perrin did not show much variations from attacks in young adults.

(2) CENTRAL PNEUMONIA.—Central pneumonia is characterized by all the symptoms of a true croupous pneumonia including leukocytosis, with a dearth of physical signs. This is frequent in children, but occurs in adults as well. I have notes of a case where the physical signs did not appear until just before the crisis, and for a day or two after the crisis were more evident than during the height of the attack.

(3) PNEUMONIA IN CHILDREN.—If a child is very young—under three years of age—the pneumonia is likely to be of the bronchopneumonic type. The child has been affected with a more or less severe bronchitis; he gradually develops fever; leukocytosis appears; the breathing rate and pulse rate increase; cyanosis is frequently seen. About this time areas of consolidation appear, frequently not involving the whole lobe, though this may occur. In older children the physical signs have all the characteristics of pneumonia in adults, except perhaps that they do not develop so early in the course.

The symptoms differ to a certain degree. In the bronchopneumonic type they are much more insidious in their beginning, though in true lobar pneumonia the symptoms in children frequently are not so abrupt in the beginning as they are in adults, though the temperature very rapidly rises. The physical signs are notoriously difficult to make out in the beginning. Perhaps the first sign is rather distant weak breathing over the affected area, to be followed in twenty-four or forty-eight hours later by slight dullness, an approach to blowing breathing, and finally typical signs of consolidation, the complete set of signs frequently not occurring until about the time of the crisis. These facts are responsible for many cases of pneumonia in children being mistaken for other conditions.

Physical Signs.—On inspection in the early stage, there is a decided limitation of motion over the affected area, provided there be a pleurisy. This is not the fact where the pleura is not affected. Later there is limitation of motion due to the lack of expansion of the pulmonary tissue. According to Osler, there is a definite increase in the size of the affected side.

Percussion early gives a high pitched tympany, or there may be a slight dullness; later the dullness merges into complete flatness over the affected area. If the pneumonia begins in the center there is little difference between the percussion note of the two sides of the chest in the beginning. Tactile fremitus is at first diminished and later increased over the affected area. It may in rare instances be less on the affected side than on the normal side.

The breath sounds in the beginning are diminished; later if there is free access of air, the breath sounds are loud and blowing and the inspiration and expiration are about equal in length and intensity. In the very early stage there may be heard the classic crepitant râle—a sound caused by the separation of the walls of the air vesicles which adhere by an exudate. It has much the sound of hair crushed between the fingers; it is heard only on inspiration. Occasionally the most marked auscultatory sign is a pleural to-and-fro friction.

Just before the crisis—sometimes as much as twenty-four hours before —a subcrepitant râle heard on inspiration and expiration can be observed over the affected area. The voice sounds are loud and seem close to the ear, giving rise to the so-called bronchophony. If the bronchus is occluded this sign may not be heard well.

As the case improves, the râles become larger and more numerous, the breath sounds become less blowing and more vesicular, and the dullness less well marked. Occasionally a lung does not resolve. Under these circumstances the dullness and blowing breath and voice sounds remain much as they were during the height of the disease. The leukocytosis as a rule does not disappear. The temperature continues high in the evening and drops to or near normal in the morning.

The diagnosis of this condition will be considered later.

Complications and Sequelae

Pleurisy.—This is such a constant accompaniment of pneumonia that its classification as a complication may be questioned. It is the pleuritic inflammation that gives rise to the pain and much of the dyspnea in pneumonia. It is recognized by a to-and-fro friction and by sharp pain on breathing. Later the visceral and parietal pleura may be separated and the friction no longer heard because of an intervening liquid. The appearance of the liquid is discerned by a marked flatness on percussion; a diminution of the breath sounds and of tactile fremitus and vocal resonance.

The dullness may be movable or the presence of the liquid may dislocate the heart or liver.

Empyema.—After the crisis the fever may again rise; or instead of a crisis the temperature may never quite fall to normal but continue as a more or less irregular type of fever—sometimes the variation is two to four degrees in twenty-four hours. In addition, leukocytosis either continues, or reappears after the count has been normal. Embarrassed breathing supervenes, the signs of liquid in the chest appear with dullness, loss of tactile fremitus and vocal resonance appear. In addition dislocation of the viscera occurs, the heart being pushed to the right or the left, depending upon which side the effusion exists.

Physical signs of effusion are so notoriously deceptive in certain cases that resort to a small aspiratory needle should be had in cases that are at all doubtful.

If the empyema is between the lobes, interlobar, the signs will be extremely deceptive. Puncture with a needle may not show the presence of liquid, either because the needle is of too small caliber, too short, or does not go in the proper direction under the circumstances. If the symptoms of fever, emaciation and leukocytosis continue, together with uncertain physical signs, and an x-ray picture shows a marked shadow, a detailed opening of the chest (done by a surgeon skilled in pneumosurgery) should be undertaken to locate the pus.

Pulmonary Abscess.—Pulmonary abscess is recognized by continuation of the fever, localized signs of dullness, loss of fremitus and voice sounds, together with pain over the region. The use of the x-ray will help much in the diagnosis. A needle may show the presence of pus, or the abscess may rupture into a bronchus.

Meningitis.—Many cases of pneumonia, especially those in which the lesion is at the apex, have severe nervous signs simulating meningitis as detailed above. In the true meningitis, however, the patient may have a convulsion, paralysis of the ocular recti and of speech—indeed, of any of the organs supplied by the cranial nerves. Opisthotonos and contractions may occur on account of inflammation of the spinal nerves.

Spinal puncture will make the diagnosis sure. If meningitis is present the spinal fluid will be more or less turbid, due to the presence of the pneumococci and possibly a complicating streptococcus or staphylococcus.

Middle Ear Disease.—Middle ear disease is a frequent and somewhat serious complication. It must be sought for by the speculum, and particularly when the patient is unconscious. It may manifest itself in conscious patients by tenderness over the tragus or over the mastoid. It is a fertile source of meningitis.

Endocarditis.—Endocarditis is recognized by pain and distress in the cardiac region, together with more rapid heart action, dilatation of the heart, and occasionally of a murmur systolic or diastolic in time, depending upon the character of the lesion produced.

Pericarditis.—This is often accompanied by very severe precordial pain, irregular heart action, dyspnea, and the presence of a to-and-fro friction synchronous with the heart action.

Jaundice.—Jaundice is rather frequently present; the jaundice usually present is probably a true obstructive jaundice due to a gastro-intestinal catarrh. In certain cases a severe hemolysis occurs which gives rise to the most intense coloring of the skin and all the mucous membranes, together with a very rapid anemia. This complication is shown by the yellow discoloration of the skin and the mucous membranes, and the presence of bile pigment in the urine.

Gangrene of the Lung.—This occurs in certain cases. The symptoms of depression increase; the lung signs clear up except over certain areas; the sputum becomes thin and extremely fetid. Often signs of a partially empty cavity can be made out over the affected area.

Gangrene of the Extremities.—This occurs occasionally—due to a thrombus of the terminal arteries. A case seen by the writer with Dr. Simcox, of Wissahickon, Philadelphia, had gangrene of the fingers of both hands. At autopsy a thrombus of the palmar arch, both deep and superficial, together with the digital arteries, was found.

Acute Dilatation of the Stomach.—Acute dilatation of the stomach often occurs as a complication or sequel of croupous pneumonia. This is recognized by collapse, distention of the abdomen—the distention being largely in the upper quadrant of the abdomen, vomiting of large amounts of fecal-like material, and obstinate constipation. It resembles obstruction of the bowels in many respects. The complete set of symptoms may be consulted under the heading of Acute Dilatation of the Stomach (p. 272).

Peritonitis.—Peritonitis occasionally occurs, and is characterized by abdominal distention, tenderness, and loss of peristalsis. This complication is rare.

Diseases to be Differentiated from Pneumonia

Pneumonia must be differentiated from:

Pleural effusion
Appendicitis and other abdominal conditions
Typhoid fever

Meningitis
Tuberculosis
Pneumothorax
Pulmonary infarcts
Pulmonary congestion
Pericardial effusion
Atelectasis
Influenza
Interlobar empyema.

Pleural Effusion.

Pleural effusion may simulate pneumonic consolidations exactly, or it may be easy to diagnose. The usual signs of effusion—flatness—which changes its level on moving the body, loss of fremitus, loss of breath sounds and dislocation of the viscera may all be present; when they are present the diagnosis is extremely easy.

Occasionally, however, the *voice* and *breath sounds* are well heard over a pleural effusion, the so-called Bacelli's sign, which may deceive one if not on guard. Fremitus is rarely present over a liquid, though it may be present in a distinctly lessened form.

If the effusion is on the left side, the heart is pushed over toward the right and its position is usually easily demonstrated by observing dislocated pulsation and percussion of the chest. If the heart is pushed to the left, however, by an effusion into the right chest, the position of the heart is best made out by palpating the apex beat. Frequently the compensatory emphysema on the left side masks the dullness, and the heart may be wrongly judged to be in a normal position. Under any of these circumstances resource to the aspiration needle should always be had.

In a pleural effusion which occurs insidiously and is not observed early, the signs may, as stated above, all resemble those of pneumonia. Here the fever may be absent, and is always less than in pneumonia. The leukocytosis is almost always less marked; the prostration is less; and in every way the patient appears less seriously ill than when he has pneumonia.

Then, too, when the dullness is in a fairly straight line, somewhat curved upward, it is likely to be due to a liquid. On the other hand, if it follows the line of either of the lobes of the lung, it is more likely pneumonia.

It must also be remembered that a pleural effusion often complicates a pneumonic consolidation. When this is the fact, liquid obtained by the needle will not exclude pneumonia. The area of paravertebral dullness is in favor of a pleural effusion though the same area of dullness can be seen in other intrathoracic conditions, as proven by Smithes.

Appendicitis.

Frequently the abdominal pain which accompanies the pleurisy complication of pneumonia is so severe, that the one complaint of the patient is this pain. The fever, the leukocytosis, the tenderness and rigidity which often are present may deceive the wisest. Great care must be taken in every case of suspected appendicitis that the chest be carefully examined. Any suspicion of lung complication will of course cause delay in opening of the abdomen.

On the other hand, severe appendiceal inflammation may cause loss of excursion and breath sounds on the right base, which will be most misleading. It is only by exercising the greatest care that these two conditions can be separated in certain cases.

Typhoid Fever.

In the early stage of pneumonia, especially where the physical signs are slight, fever may be the only symptom. Here a leukocytosis would at once mark the disease as at least not uncomplicated typhoid.

Many cases of central pneumonia with fever as the chief symptom are mistaken for typhoid until a crisis shows the error. Usually careful examination of the chest will point to the diagnosis. The presence of a Widal reaction and the absence of a leukocytosis will point toward typhoid fever.

Error may occur in cases of typhoid fever with pulmonary complications. In these cases the Widal reaction or a blood culture will make a diagnosis of typhoid fever as the underlying cause.

Meningitis.

Meningitis is marked by severe cerebral symptoms: headache, photophobia, stiffness of the neck, Kernig's and Babinski reflexes, and paralysis of the ocular muscles. There is no lung involvement in meningitis not due to pneumonia or tuberculosis. In certain cases of pneumonia, however, with slight lung involvement, there may be great difficulty in making a diagnosis. Spinal puncture in these cases will show whether there is a true meningitis and what the infecting organism is.

Tuberculosis.

In tuberculosis there may be a true pneumonic consolidation which has its origin about an old tubercular lesion. The cause of the outbreak may be either a new growth of tubercle bacilli, or a true pneumococcic inflammation. Such attacks are likely to be mistaken for an ordinary pneumonia, and the differentiation is often very difficult. The difficulty increases when the condition is a simple bronchopneumonia. In tuberculosis, however, the course is atypical. The onset is usually more gradual; the expectoration is likely to be pure blood. The case does not end in a sharp crisis, or in a short lysis; instead, the fever lasts for days and is of an irregular type.

The physical signs differ in that there is usually not the same sharply outlined blowing, breathing and bronchophony common in uncomplicated croupous pneumonia; the breathing is more distant—more of a type one hears over an ordinary tubercular infiltration. Tubercle bacilli may often be found in the sputum. McCombs reports a case where these were found as early as the seventh day.

The final outcome is different, the tubercular cases running into chronic tuberculosis when they survive the attack; while the simple croupous pneumonia entirely recovers.

Pneumothorax.

Pneumothorax might be mistaken for pneumonia, but the onset is more sudden; the physical signs are entirely different. There is usually tympany, amphoric breathing or absent breath sounds and voice sounds with loss of tactile fremitus and dislocation of the viscera in pneumothorax, which does not occur in pneumonia. In certain cases of pneumothorax where the air is under great tension the percussion note is *dull* instead of being tympanitic.

Pulmonary Infarct.

This is of sudden onset; there is frequently expectoration of pure blood; there is often collapse at first. The condition accompanies some septic process or is the result of cardiac disease. Later there are all the physical signs of pneumonia, but they rarely embrace the entire lobe of a lung.

Pulmonary Congestion.

Pulmonary congestion sometimes occurs acutely. There is dullness, distant breathing and a few râles over the area affected. The symptoms are much milder than in pneumonia and they all disappear in twenty-four to forty-eight hours. Congestion, however, the result of cardiac disease or a weakened heart from prolonged infection, may have slight fever with leukocytosis. The dullness is apt to be at both bases, but may affect only one. The dullness is less marked than in pneumonia and the râles are larger and more moist.

Pulmonary Atelectasis.

Pulmonary atelectasis occurs most frequently in children. It usually

TABLE OF DIFFERENTIATION

	Onset	*Blood*	*Symptoms*	*Nervous Symptoms*	*Abdominal Symptoms*	*Fever*	*Spinal Fluid*	*Expectoration*	*Physical Signs*
Pneumonia..........	Rapid	Leukocytosis	Chill. Fever Dyspnea Chest Pain	May occur; not constant	Occur in certain cases	High; abrupt beginning and ending 6 to 9 days	No change; unless meningitis present	Blood stained; rusty	Dullness; blowing breathing; increased voice sounds
Pleural Effusion....	Gradual	Slight leukocytosis	Insidious	None	Rare	Slight	No change	None	Dullness Diminished voice and breath sounds Diminished fremitus
Appendicitis..........	Sudden	Leukocytosis	Abdominal pain Rigidity Tenderness	None or rare	Marked	High or moderate	No change	None	Abdominal tenderness and resistance
Typhoid Fever.......	Gradual	Leukopenia	Fever Delirium Diarrhea	Constant	Often marked	Gradual beginning; lasts 3 or 4 weeks	No change	None or mucous	Abdominal distension Rose spots
Meningitis...........	Rapid or gradual	Marked leukocytosis	Nervous system Convulsions	Marked	None	Irregular	Cloudy; shows organism	None	Change in reflexes
Tuberculosis.........	Gradual	Leukopenia	Irregular	None	None	Irregular	No change	Pure blood; tubercle bacilli are found in sputum	Chest signs; dullness Increased voice and breath râles
Pulmonary Infarct. .	Sudden	Leukocytosis	Sudden collapse Dyspnea Pain in side	None	None	Slight and irregular	No change	Pure blood	Dull over small areas of chest
Pulmonary Congestion...............	Gradual; sometimes abrupt	No change, or slight leukocytosis	Dyspnea Slight fever	None	None	Slight	No change	None or slight	Dull over basis of chest
Atelectasis..........	Sudden	No change	Dyspnea	None	None	None or slight	No change	None	Dull over small chest areas
Interlobar Empyema.	Gradual	Leukocytosis	Fever Irregular emaciation Dyspnea Cough	None	None	Irregular	No change	None or slight	Dullness. Loss of breath and voice sounds often over lines of lobe division

affects small areas. The physical signs are dullness and distant breathing. If the area affected is large, there may be a distinct cyanosis.

INFLUENZA.

Influenza may be confused with pneumonia; the mistake is more likely the failure to distinguish a pneumonia.

Uncomplicated influenza does not give a leukocytosis. When a case of influenza has a sudden or very prolonged fever accompanied by leukocytosis, careful search must be made for consolidation of the lung. During epidemics of influenza, the general practitioner is apt to be so overworked that he fails to make a careful physical examination. Attention to the above points will obviate the error.

INTERLOBAR EMPYEMA.

It is most important to differentiate this from pneumonia. Frequently it follows an attack of pneumonia, and is erroneously thought to be an unresolved pneumonia. It *should always be remembered that fever of intermittent type, following pneumonia, with prevalence of the physical signs to a greater or lesser degree is most likely to be due to a collection of pus—either free in the pleural cavity or confined between the lobes—interlobar empyema (though tuberculosis must also be remembered).*

The symptoms are irregular fever, continued prostration, emaciation and leukocytosis. There is dullness, distant breathing, and diminished or entire loss of fremitus.

Demonstration of pus by the needle or by operation is the positive sign of empyema.

7. Cerebrospinal Fever

(*Cerebrospinal Meningitis, Spotted Fever*)

Organism.—This complaint is due to the entrance into the body of the organism, Diplococcus intracellularis meningitidis of Weichselbaum. The portal of entry is apparently the nasopharyngeal mucous membrane.

Flexner has proven by experimentation on monkeys that if the organism in pure culture is introduced into the nasal mucous membrane, the animal, after a few days' sickness, develops typical symptoms of the disease, and at post mortem the lesion of the malady is found in the spinal cord. It is probably an air-borne disease, though the exact means of transmission are not yet known.

The pathognomonic lesion is found in the meninges of the brain and the spinal cord. There is cellular infiltration of the pia. These infiltrating cells which are usually polymorphonuclear in character, contain the organism.

Exudate—Spinal Fluid.—The exudate varies from a simple purulent contamination to a thick layer of new membrane covering the entire cord. The spinal fluid is in the great majority of cases opalescent, due to the presence of many polymorphonuclear leukocytes which contain the diplococcus. In cases which die late, the exudate is fibrous in character and is found to compress the surrounding nervous tissues, until they cease to functionate.

In severe cases the kidneys show acute inflammatory changes, as does the heart muscle.

General Symptoms.—The disease is usually ushered in with sudden, severe, almost unbearable headache, with fever and vomiting. Occasionally the attack begins with convulsions. This is particularly likely to be the fact in children. In the course of a short time, varying from twelve hours to two days, the typical signs of meningitis occur. Stiffness of the muscles of the neck is the first of these to appear. This in severe cases rapidly develops, until the entire group of muscles of the back are involved, and the child is in a state of opisthotonos—the back is arched. If the child be taken by the head, the entire body can be lifted from the bed as though the spinal column were one inflexible piece. The thumbs are drawn into the palms, and the arms become spastic.

Although Kernig's sign is not pathognomonic, it rapidly develops in many cases. If the thigh be brought at right angles to the body, and an attempt be made to extend the leg upon the thigh, the extension of the leg will be prevented by contraction of the muscles. The point to which the flexed leg varies, is between a right angle of the leg to the thigh, and an angle of 135 degrees. If an attempt be made to straighten the leg beyond that point, the patient evinces great pains.

Babinski's sign—the extension of the great toe when the inner side of the sole of the foot is scratched—is of frequent occurrence.

Brudinski's reflex is frequently present. It consists in the fact that if one leg is passively flexed, the opposite one is coincidently flexed (identical collateral reflex), or if the leg is passively flexed in certain cases, the opposite leg is extended instead of flexed (reciprocal collateral reflex).

The patient soon becomes delirious; thereupon the mind becomes clouded, and the patient rapidly becomes unconscious. Frequently before unconsciousness supervenes, strabismus occurs because the nerve supply of ocular muscles is involved. The pupils also become unequal and fixed.

Herpes of the lips occurs in many cases.

Hyperesthesia is common, the individual being disturbed by the least touch. Often the patient will lie quietly coiled up; if he is disturbed, he becomes querulous and immediately resumes his position, much as an individual does who is recovering from a cerebral concussion.

Special Symptoms.—The Rash.—A petechial eruption, occurring first on the abdomen, occurs in certain cases. The rash is a true petechia—

fire-bright red or bluish, about the size of a flea bite—later becoming larger and affecting any portion of the body. It makes its appearance on the third day. It is not as common as the name "spotted fever" would indicate.

Fever.—Fever is a constant symptom, ranging from 101° to 104° or 105° F.

The Blood.—*Leukocytosis.*—There is always a leukocytosis of polymorphonuclear type, ranging from 16,000 to 40,000 to the cm.

The spinal fluid is turbid, the turbidity being due to the presence of leukocytes. The leukocytes on being stained with Wright's stain, or by Gram's method, are found to contain the characteristic Gram-negative diplococci. Indeed, the presence of the **diplococci** in the leukocytes of the spinal fluid is the only certain means we have of differentiating this form of meningitis from other forms which closely resemble it.

Occasionally the spinal fluid is not turbid, as in one particular case of the author's. This was a typical case of cerebrospinal fever, as proven by all the symptoms. The cord was tapped with the individual lying on the side; the fluid was clear and there was a scarcity of leukocytes. It is suggested that this error might be obviated if the tapping were done with the patient sitting up.

Diseases to be Differentiated from Cerebrospinal Fever

This disease must be separated from:

All of the infectious fevers
Scarlet fever
Measles
Pneumonia
Typhoid fever
Simple attack of indigestion
Various other forms of meningitis
Anterior poliomyelitis.

Scarlet Fever.

Scarlet fever begins much as does cerebrospinal fever, with sudden high fever and often convulsions, but in scarlet fever there is the characteristic *sore throat;* the *red congested pharynx and fauces.* The rash occurs within the first twenty-four hours and is not large, discrete and petechial, but small, thickset, red, discrete spots, between which there is a scarlet erythema. In scarlet fever there is no herpes; there is leukocytosis, and a perfectly clear spinal fluid, except in those rare cases where a streptococcic meningitis exists as a complication of scarlet fever. There are more of the signs of involvement of the cord, such as stiffness of the neck, strabismus, etc.

MEASLES.

Measles is differentiated from cerebrospinal fever by the appearance of coryza, bronchitis, Koplik's spots and the typical crescentic rash of measles, the latter occurring on the third day, appearing first on the face. The spinal fluid of measles is clear and does not show diplococci.

PNEUMONIA.

In pneumonia there is the presence of the physical signs in the chest, dullness, increase of breath and voice sounds over the affected area in the very early stages. Pneumonia has herpes and leukocytosis to confuse. It often, especially in children, begins with convulsions, and in certain cases where the meninges are affected, either by an actual pneumococcic meningitis or by a toxic condition, spinal puncture must be resorted to to settle the diagnosis. Here if there is a true pneumococcic meningitis, there will be found diplococci in the spinal fluid; but the fluid is usually only slightly turbid, the diplococci are usually outside the cells and the few cells present are found in the fluid.

TYPHOID FEVER.

Some cases of cerebrospinal fever closely resemble typhoid fever in their beginning. The onset is gradual; the meningeal signs are rather those of a meningeal form of typhoid fever than those of a true meningitis. However, the regular course of the temperature range in typhoid fever, the enlargement of the spleen, the diarrhea, the Widal reaction and the absence of leukocytosis will differentiate the cases. If there is still doubt, the spinal fluid will decide the question. The spinal fluid in typhoid fever is clear, while that of cerebrospinal fever is cloudy.

INDIGESTION.

Certain cases have only the characteristics of an indigestion as evidenced by vomiting and convulsions, but a simple indigestion does not have a leukocytosis; it does not have continued fever, and further, the spinal puncture gives a negative finding.

TUBERCULAR AND OTHER FORMS OF MENINGITIS.

From tubercular and other forms of meningitis, cerebrospinal fever can be surely differentiated only, by spinal puncture, and the finding of polymorphonuclear leukocytes containing a Gram-negative diplococcus. However, the temperature of the tubercular cases is much more apt to be irregular. There are paralyses—especially about the face—which may be fleeting in character. The pupils are apt to be irregular or they may not react to light.

The fluid of tubercular meningitis is clear; it contains a few leukocytes which are largely lymphocytes, and if it is collected under sterile precautions and centrifugated for several hours the tubercle bacilli can usually be demonstrated. One case of turbid fluid obtained in a case of Streptococcus meningitis, due to middle ear disease, contained streptococci in the fluid and a few in the cells. This simple procedure of tapping the spinal cord is easily learned, and should be practiced by every physician.

Spinal puncture should be undertaken whenever there are cerebrospinal symptoms sufficient to suggest meningitis—often it is the only procedure which will establish a diagnosis.

Anterior Poliomyelitis.

Certain cases of this condition might in the beginning be taken for cerebrospinal fever, owing to the presence of convulsions and rigidity in certain cases, but if the case lives a flaccid paralysis supervenes. Spinal puncture will reveal a clear fluid.

The following from an article published for the author in Archivis Diagnosis, April, 1908, will be of value:

While spinal puncture is of course not to be practiced without thought, it is a method well within the province of the man doing general practice.

To be of value, the physician practicing this diagnostic help must first know how to perform the slight operation and then to properly examine the fluid obtained. The latter presupposes the possession of a microscope and knowledge sufficient to use it. This knowledge is common to all recent graduates and can be obtained by all of us by the perseverance in a little hard study. This much we owe to our patients.

Technic of the Operation.—The only instrument necessary is a sharp hollow needle, three or four inches long and a little smaller than an ordinary match stick, with an obturating stillet beveled at the end on a level with the point of the needle. The operation should be performed under strict asepsis.

The needle is boiled. The lumbar and sacral region of the patient's back are scrubbed with soap and water, washed with bichlorid solution and finally with alcohol or iodin may be used. The operator's hands should be scrubbed and thoroughly soaked in bichlorid solution and then in alcohol. These preparations being completed, the patient is laid on either side, the spinal column being bowed forward as much as possible. The point of puncture is one-half to one-quarter inch to one or the other side of the line of the spinous processes and between the second and third or third and fourth lumbar vertebrae. The needle is held directly perpendicular to the spinal column, and is plunged through the skin with a quick motion. The point of the needle is then directed slightly upward and toward the median line. If the aperture between the bodies is reached the needle is felt to slip into the spinal canal and the fluid at once begins to flow. If the needle impinges against the body of the vertebrae it is pushed gently in all directions until the aperture is found. If there is much bleeding and the fluid does not flow, the wire which has been boiled with the needle should be pushed into the caliber and a possible blood clot displaced. The operation is but slightly painful. It should be performed in all cases where the symptoms and physical signs point to a probable meningitis. If so performed and the fluid properly examined the operation will frequently clear up a diagnosis and point to a proper treatment; it will take but a short time. I have frequently performed the operation, found a normal spinal fluid, and the patient recovered none the worse for the procedure.

The following abstract of cases will serve to make my points clear:

Case I.—John A., aged four years, was admitted to St. Timothy's Hospital suffering from what was supposed to be typhoid fever. The child, however, had been taken suddenly ill with high fever and rather marked unconsciousness. On admission he had a temperature of 103° F., was unconscious, and had a stiff neck, a well-marked Kernig sign and a tache. Spinal puncture was made, a very slightly turbid fluid was removed. This was centrifugated and diplococci lying *outside* of the cells were demonstrated. Careful examination of the lungs showed small areas of consolidation. The child died and areas of consolidated lung were found, together with an intense cerebrospinal meningitis. Pneumococci were demonstrated in both the meninges and the lungs.

Case II.—A boy, aged seven, was taken suddenly ill with fever and delirium while away at boarding school. On admission to St. Timothy's Hospital he was found to have the physical signs of meningitis with fever and delirium. A puncture was at once made. The fluid was opalescent, and on being centrifugated for a few minutes a large precipitate was found. Under a one-twelfth oil immersion this precipitate was found to be composed largely of polymorphonuclear leukocytes, and to contain from one to six diplococci in many of the cells. The case was clearly one of the epidemic form of meningitis and was isolated. Within a few days two other exactly similar cases were admitted and diagnosed in the same manner.

Case III. (seen with Dr. Devitt).—C. R., aged five years, was suddenly seized with vomiting. There was no fever. The bowels were loose. He vomited for two days and nights. I saw him on the third day, when he was slightly dull, had a distinct tache, but no other signs of meningitis. Two days later, there was slight strabismus with a temperature of 100° F. A spinal puncture showed a perfectly clear fluid. This was collected in sterile centrifuge tubes, and centrifugated in those tubes at a high speed for twelve hours. There was a very slight sediment containing a few lymphocytes. It was stained for tubercle bacilli and after a few minutes beautifully stained tubercle bacilli were found lying in a perfectly clear field. I think that the direct collection of the spinal fluid into sterile tubes is of great value. Previously when centrifugating was practiced for a number of hours and the tubes had not been made sterile, there was so much growth of contaminating bacteria, that an accurate observation was difficult.

Case IV.—Another case worth recording was that of a young woman, who was brought to St. Mary's Hospital with the history of a long siege of nursing, followed by headache for a day and sudden delirium. When brought to the hospital she was restless, semi-conscious, had a tache, no fever, and appeared to be hysterical. In a day or two she developed a fever and a nondescript rash (this latter proved to be due to atropin). A puncture was made; a clear, liquid free from germs of any kind was found. The case drifted into a demented condition. The puncture proved no meningitis.

Case V. (seen with Dr. Eichman).—A boy, aged four years, had a suppurating middle ear and a severe nephritis following scarlet fever. He was seized in the middle of the night with severe convulsions and high fever. These attacks were taken to be due to the uremic poisoning. In hope of relieving the convulsions, which were uncontrollable, a spinal puncture was made. A turbid fluid was thereupon withdrawn, which on being centrifugated showed many polymorphonuclear cells, and very numerous cocci, some free, some within the cells. The convulsions and fever continued until death. To our surprise the child proved to have meningitis, which in all probability was the cause of the convulsions.

These cases tend to demonstrate a few of the points of value of a spinal puncture. The first case might readily have been mistaken for epidemic cerebrospinal meningitis. In fact it was a pneumococcus infection. The second, so far as symptoms proved, was similar to the first, but the puncture showed the presence of the epidemic disease. The procedure in the third case was of great value, because it made certain a diagnosis of tubercular meningitis in a doubtful case. The fourth case was uncertain from the beginning, and the puncture showed at least that the patient did not have meningitis. I feel quite sure that no one would have thought of meningitis in the last case without a puncture. As a matter of fact, the puncture was performed as a therapeutic measure, but proved of diagnostic value. These cases all go to show the great diagnostic value of the procedure. Two of them were done on the ordinary routine of private practice.

8. Influenza

Organism.—Influenza is an acute infectious disease due to implantation of the Pfeiffer bacillus or influenza bacillus.

Symptoms.—This disease is characterized in typical cases by sudden fever, aching in the extremities, coryza, sore throat and cough. There is usually much prostration. One curious characteristic the writer has noticed is soreness of the scalp, so that if the hair is slightly brushed or slightly pulled, the patient manifests discomfort because of the pain developed. The fever which accompanies this condition usually rises suddenly; in cases where there is some complication it is exceptionally high, running a course of three, four, to seven days. Frequently there is much bronchitis and much discharge from the throat and nose. There is a tendency to neuritis, to diseases of the middle ear, to accessory sinus disease and to pneumonia.

The disease is spread like "wildfire" over a community; often all the members of a family are stricken at one time or within a very few days. There is often severe diarrhea and vomiting. Frequently the patient, if he is young or aged, or for any reason is below par in general condition, becomes stuporous and delirious. Occasionally the heart is extremely weak in this same class of cases. In the same epidemic, even in the same family at the same time special organs appear to bear the chief burden of the disease.

The respiratory organs are those most commonly affected—indeed, in practically all cases there is a respiratory infection present. In the respiratory type, however, the respiratory passages, the nose, the throat, larynx, trachea, bronchi and lungs are all inflamed. There is great prostration. When the rhinitis is severe, the accessory sinuses are particularly likely to be inflamed, the sinusitis remaining for a long time after the case is cleared up. The tonsils are often inflamed, and the cultures show the presence of influenza bacilli. Marked hoarseness amounting to a true aphasia often occurs when the larynx is much affected. In the first 24 or 48 hours, sometimes in the very beginning, there is a more or less severe bronchitis which becomes the dominating feature of the attack.

Bronchopneumonia is a common sequel or complication of the disease, the fever rising, leukocytosis supervening, and the case taking on the physical signs of consolidation, irregularly situated, and of greater or less extent.

Abscess and gangrene of the lung have been reported as the sequel to the pulmonary form of the disease.

In the *nervous type* of cases there may be a *severe neuritis;* there may be *delirium, coma* or *convulsion.* Following the attacks, *pain along the course of the nerves* is extremely common.

A true *meningitis* may occur. Flexner has described such cases in which influenza bacilli have been found.

The *gastro-intestinal tract* seems to bear the brunt of the attack in certain cases—*vomiting, diarrhea, gall-bladder symptoms* predominating.

Diagnosis.—The diagnosis must be made first, by the presence of an epidemic of the character described, and second, by finding the Pfeiffer bacillus in the nasal discharge or in the sputum. In doubtful cases it must be cultivated. It is a habit since 1889, when influenza was extremely common in the United States, to call every cold "influenza." It is quite probable that many of the attacks of infectious colds are in truth influenza, but it seems to the writer that unless the diagnosis be proven by a bacteriological examination, such attacks should be called infectious coryza, bronchitis, etc., rather than influenza.

Diseases to be Differentiated from Influenza

It must be distinguished from:

Infectious colds
Tonsillitis, and in the beginning from
Typhoid fever
Pneumonia
Tuberculosis.

TONSILLITIS.

Tonsillitis, due to staphylococcus or streptococcus, may resemble influenza in the aching of limbs and the high fever, but it is distinguished by the absence of symptoms of coryza, of bronchitis, and by the presence of a distinct exudate in the throat. Occasionally influenza may be mistaken for diphtheria, just as it may be mistaken for tonsillitis, but a bacteriological examination of the throat contents will make the diagnosis certain. If influenza bacilli are present in the culture, positive for other organisms, there is a double infection present.

TYPHOID FEVER.

Typhoid fever rarely begins abruptly as does influenza, but may do so. In typhoid fever the tongue is quite characteristic; there is less coryza and less bronchitis. There is more abdominal distention. In cases where the main brunt of the inflammation in influenza is borne by the gastro-intestinal mucous membrane, the disease is especially difficult to distinguish from typhoid fever. In typhoid fever leukopenia exists, whereas in influenza the leukocytes are normal in number, or very slightly increased. The presence of Widal reaction with typhoid of course marks the case as one of typhoid fever. The final course of the disease also makes a certain diagnosis, and a blood culture would show a growth of the particular organism infecting the patient.

PNEUMONIA.

The beginning of pneumonia is instanced by increased respiratory trouble, by leukocytosis and beginning of dullness in various parts of the lung. The pneumonia may be of a bronchopneumonic type, the case running on for a time, but physical examination and blood examination will usually make the diagnosis.

If a meningitis occurs there will be delirium, headache, and opisthotonos and the diagnosis must be proven by spinal puncture. Flexner has recently called attention to influenza meningitis where the causative factor was the influenza bacillus.

TUBERCULOSIS.

Tuberculosis, from the beginning, has constantly been mistaken for influenza. The reason for this lies probably in the fact that tuberculosis which has been quiescent is very apt to be relighted by any acute infection. Here consolidation of the lung, the presence of tubercle bacilli in the sputum and the history of the case should enable one to make the diagnosis.

9. Whooping-cough

(*Pertussis*)

Organism.—The organism described by Bordet and Gengou, often known as the Bordet bacillus, is regarded as the cause.

Symptoms.—Whooping-cough is an infectious disease, characterized by severe paroxysmal attacks of coughing of laryngeal character, usually worse at night.

Duration.—The duration of the disease is from nine to twelve weeks.

Course of the Disease.—The disease usually passes through three distinct stages. The first *catarrhal stage* is marked by a bronchitis resembling an ordinary case of bronchitis, with this difference perhaps: the cough present seems to be out of proportion to the physical signs, and often in this stage the attack of paroxysmal cough can easily be brought on by excitement, crying, laughing, or overexertion. It lasts from one to two weeks, and merges into the *paroxysmal stage.* Here the cough comes on in spells. Each attack of coughing begins with three or four short, sharp, explosive attempts to cough in order to rid the larynx of an irritation, followed by a long drawn, more or less crowing inspiration—the so-called whoop. If the case is entirely uncomplicated the child is well between the paroxysms of coughing, there being no bronchitis, fever, or other untoward symptoms.

Frequently the child vomits during a paroxysm. The attack of vomiting usually terminates the paroxysm. Occasionally the local congestion of the face, head, and nose is so severe, that the countenance is perfectly livid; blood may drip from the nose, and subconjunctival hemorrhage occur. Always the inability of the child to inspire causes the face to flush, even though the obstruction be not so severe as to cause cyanosis. The number of paroxysms vary from ten to fifty in twenty-four hours.

In the beginning the disease is difficult to differentiate by the cough itself from attacks of cough due to bronchitis, so common to children. However, there is usually freedom from bronchitis; the cough is worse at night; exertion or excitement are fairly sure to bring on an attack of coughing.

This paroxysmal stage lasts from four to six weeks and is followed by the *stage of decline,* during which the paroxysms become less frequent, the child reaching a normal condition in from two to three weeks. Attacks of paroxysmal cough, however, may last much longer than this.

The various organs are but little affected in uncomplicated cases. The disease kills through serious and frequent complications rather than from its own virulence, though in very young children the attack may cause a fatal syncope or such a severe interference with the circulation that a convulsion may be fatal.

Diagnosis.—Differential count of the blood shows a lymphocytosis, varying from 50 per cent to 85 per cent. This is of the greatest value in enabling one to arrive at a diagnosis before the typical whoop occurs.

Rurah describes the following method of ascertaining the whoop in uncertain cases. The child is held as in examination of the throat; then the tongue depressor is pushed far back until the epiglottis is reached. This will bring on a paroxysm of coughing much more severe when the condition is whooping-cough than in any other condition.

The fact that whooping-cough is highly contagious and that many children are suffering from it in the neighborhood, should at once brand any spasmodic cough which is worse at night, as probably one of whooping-cough.

Complication.—*Pneumonia,* usually of the bronchopneumonic type, is a common and frequently fatal complication.

Diseases to be Differentiated from Whooping-cough

Whooping-cough must be distinguished from:

Bronchitis, laryngitis, laryngismus stridulus, obstructive laryngitis, foreign body in throat, pressure by enlarged lymphatic glands or of the thymus gland, from paralysis of the recurrent laryngeal nerve and from intratracheal growths.

BRONCHITIS.

Bronchitis may complicate whooping-cough. If it does there is difficulty in making a differential diagnosis between a primary bronchitis and one complicating whooping-cough, but if there is a lymphatic leukocytosis and if the typical whoop develops one may be sure of the diagnosis of whooping-cough even before the whoop occurs.

LARYNGITIS.

Laryngitis, whether it be of a simple catarrhal variety or diphtheritic, or due to the presence of a foreign growth, gives rise to a spasmodic cough of laryngeal character. However, there is lacking the characteristic whoop, which is preceded by the several spasmodic expiratory coughs. Examination of the blood, as stated above, will show the absence of a lymphocytosis.

LARYNGITIS STRIDULUS.

Laryngismus stridulus is almost without exception one of the expressions of rachitis or tetany, and the inspiratory "crowing" sound follows a spasmodic closure of the glottis and not a paroxysm of coughing.

FOREIGN BODIES.

Foreign bodies may cause a spasmodic cough not unlike whooping-cough. There is usually a history of an inspired body. The symptoms begin immediately. There is no lymphocytosis. The x-ray will show certain foreign bodies in the larynx or bronchi.

Enlargement of the peribronchial gland or the thymus gland is very frequently accompanied by a cough not unlike that of pertussis. Careful examination will usually reveal first the absence of the history of exposure to whooping-cough. The paroxysms lack the inspiratory whoop. The cough is not more frequent at night than during the day. There is a more or less constant retraction of the suprasternal notch and epigastric angle. When the stethoscope is placed over the trachea, high pitched inspiratory and expiratory sounds will indicate compression of the trachea or bronchi. Percussion over the interscapular space and under the sternum will show dullness. If the thymus is involved, there will be dullness anteriorly rather than posteriorly. The x-ray will show a shadow either in the position of the thymus, or along the bronchi, indicating enlarged glands.

Lymphocytosis is not such a valuable sign of distinction, because while the lymphocytes are increased to a greater extent in whooping-cough, they are increased to a certain extent in glandular enlargement.

PARALYSIS OF THE RECURRENT LARYNGEAL NERVE.

This will give rise to a paroxysmal cough, but there is usually not a distinct whoop. Laryngeal examination with a mirror will show a paralysis of one of the vocal cords.

10. Gonococcic Infection

This article will not deal with the local neisserian infection of the genital organs, but with a general gonococcic bacteriemia, with its local manifestations in the eye, in the heart and in the joints.

Gonococcic Ophthalmia

General Statements.—The disease is highly contagious when any of the gonococci are found in pus of the ophthalmia. In children gonorrheal ophthalmia is practically always secondary to a local gonorrheal lesion in the genital organs, either of the parents or of the nurse.

Gonococci ophthalmia occurs in both adults and children. It is much more common in the newborn, the infection taking place at the birth of the child by contamination from the vulva and vagina of the mother or from some accidental contamination.

The first symptom is redness of the conjunctiva of one or both eyes, followed by purulent inflammation and by great swelling of the eyelid. The conjunctiva becomes beef-red in appearance and edematous; this chemosis frequently makes any view of the cornea impossible. If the condition be not early relieved, the cornea rapidly becomes cloudy, swollen and ulcerated, so that upon healing the cornea is the seat of a scar, and the sight of the child is more or less impaired—frequently the sight is entirely lost.

The case proceeds from a mere redness of the eye to entire destruction of the entire eyeball with terrifying rapidity. There are milder cases, where for some reason, either the lack of virulence in the organism itself or in the resistance of the child, destruction of the eyeball does not take place.

Diagnosis.—This must be distinguished from *inflammation* of the conjunctiva, nonspecific in origin. The diagnosis is best made by staining a specimen of the pus from the exudate. If the case is gonorrheal in origin, the gonococci will be found within the cells in the pus forming the exudate. If no such gonococci are found, it is fair to suppose that the condition is nonspecific in character. Nonspecific lesions are not nearly so virulent in their progress, and practically never destroy the sight of the eye. While this diagnosis is easily made, time must never be lost, but the proper treatment to combat gonorrheal inflammation must be instituted when there is the least doubt as to the character of the infection.

Gonococcic Endocarditis

Symptoms.—This condition may be suspected when symptoms of inflammation of the valves of the heart appear in the course of an attack of gonorrhea. The same remark may be made of pericarditis. Both of these conditions, endocarditis and pericarditis, may be characterized by the appearance of fever and increased leukocytosis plus physical signs referable to either the heart valves or the pericardium—namely, adventitious sounds in the heart area.

Diagnosis.—The symptoms of endocarditis arising from gonorrhea do not differ from endocarditis of any other origin, except perhaps they are more apt to be of a malignant character. The reader is referred to the chapter on Endocarditis for the diagnosis.

Gonococcic Arthritis

Inflammation of the joints is an extremely common occurrence in gonorrhea.

Symptoms.—It is usually nonarticular as contrasted with the arthritis of rheumatism, which is mostly polyarticular. In the course of an attack, a joint suddenly becomes swollen, red and painful; the inflammation persists in the joint and frequently suppuration occurs, and the functional activity of the joint is destroyed or impaired. There is always fever which is of a septic type. Occasionally more than one joint is involved, a true polyarthritis taking place.

Diseases to be Differentiated from Gonococcic Infections

The condition must be differentiated from:

Traumatism
Rheumatism
Other septic arthritides.

TRAUMATISM.

This diagnosis is simple because there is usually a history of an injury. One precaution is necessary if the case is at all doubtful—the part should be examined by means of the x-ray—for in many instances there may be no history; the case may closely resemble rheumatism of the nonarticular variety; and the skiagraph shows a fracture or other evidence of injury.

RHEUMATISM.

Rheumatism is usually polyarticular; the inflammation flits from one joint to another; a joint affected today will be well tomorrow. There is usually but little exudate, and that is serous and not purulent in character; there is no destruction of the articular surfaces; the fever is more continuous and less septic in type than in gonorrheal arthritis.

GONOCOCCEMIA.

This condition is the result of the presence of gonococci in the general blood stream. The condition is one of the most serious found. There is fever of the septic type ranging from normal to 103° or 104° F.; rigors are frequent, the temperature rising rapidly after the chill. The patient rapidly emaciates; there is leukocytosis; frequently foci of suppuration develop in different parts of the body, and the condition of pyemia occurs. In this state the patient may be thought to be suffering from typhoid fever, but the presence of leukocytosis and absence of a Widal reaction, the inflamed joints and the general septic condition of the patient, and lastly the recovery of gonococci from the blood, make the diagnosis certain.

Two important means are now employed to *differentiate gonorrheal infections from those by other organisms*—the complement fixation test and the reactive test by the injection of culture of killed gonococci. (The reader is referred to the excellent article by Schwartz and O'Neill, Am. J. of Med. Sci., 1911, CXLI, p. 693; also 1912, CXLIV, pp. 369-815, for details of the procedure.) According to Cole, injections of the killed gonococci give both a local and general reaction, and a vaccination by a glycerin extract (gonococcin) will give a local reaction resembling that observed in the von Pirquet reaction for tuberculosis in tuberculous individuals.

PROCTITIS.

Proctitis occurs in rather a large number of cases of gonorrhea, sometimes acute, sometimes chronic. In cases of proctitis, even with no history of gonorrhea, one of the more recent tests should be used in order to endeavor to discover the etiology.

GONORRHEAL STOMATITIS.

This occurs in certain cases. It is constantly mistaken for stomatitis due to other conditions. The diagnosis can be made by its association with genital gonorrhea, and with gonorrhea in the mother of the child, or above all by the *cultivation of the gonococci directly from the lesion.*

PHLEBITIS.

Phlebitis may be the result of gonococcic infection, and in doubtful cases careful examination of the other organs, its association with other lesions of gonorrhea, and finally the complement fixation and vaccination test will help to make the diagnosis.

11. Bacillary Dysentery

Organism.—Observations of Shiga called attention to the fact that much of the tropical dysentery was due to the specific action of a bacillus which has been called by the observer's name—otherwise Bacillus dysenteriæ.

Since Shiga's observations, Flexner, Duval, Hunt and others have proven that most of the attacks of dysentery throughout the entire country are due to the presence of various strains of this same bacillus.

Symptoms.—The symptoms of the disease, as seen in the temperate zone, are sudden abdominal pain, vomiting, fever, diarrhea and prostration. The attack often begins with a copious watery diarrhea, soon to be converted into frequent, mucous, bloody stools with much tenesmus and with persistent vomiting.

Course of the Disease.—Occasionally attacks begin with a colitis as the prominent symptom. In severe cases and in fatal cases, delirium supervenes, and the patient passes into a typhoid state, and may die in three or four days. In the vast majority of cases the patient gradually improves under proper treatment, and while he is much prostrated, eventually recovers.

In the tropical and subtropical regions, the symptoms are much more severe, and the mortality is higher.

Complication.—*Nephritis* is a common complication.

Diseases to be Differentiated from Bacillary Dysentery

This disease is to be differentiated from:

Appendicitis
Ordinary diarrhea

Ovarian cysts
Hemorrhoids and polypi
Amebic dysentery
Enteritis
Poisoning by corrosive chemicals
Pressure in the pelvis
Cholera.

APPENDICITIS.

Certain attacks of appendicitis begin with an actual diarrhea or dysentery as an initial symptom, but this is rare, and without exception the local condition may be made out by tenderness, by resistance and by recurring pain in the appendiceal region. There is practically always a leukocytosis in appendicitis.

ORDINARY DIARRHEA.

Ordinary diarrhea has as its exciting cause some error in diet. The stools are large and watery, but are rarely dysenteric. With the correction of diet and rest such cases always get well. In certain simple cases there is *colitis* which brings about small stools with tenesmus. The difficulty of differentiating this from bacillary dysentery can be overcome by taking a careful history, by performing the agglutination reaction with the serum of the patient's blood and different stains of Bacillus dysenteriæ and by cultivation of the bacillus from the stools. The latter of course can only be done in a well-equipped bacteriological laboratory. Simple colitis is not dependent upon Bacillus dysenteriæ.

OVARIAN CYSTS.

Ovarian cysts, particularly dermoids and other growths in the pelvis, may bring about such an irritation of the colon that it amounts to an actual inflammation, and on account of the frequent mucous stools with tenesmus and blood, be mistaken for dysentery. A diagnosis can be made by a careful physical examination of the pelvis and by blood tests for the agglutination reaction with dysentery bacilli.

HEMORRHOIDS AND POLYPI.

Hemorrhoids, polypi and cancer of the rectum may simulate dysentery from the fact that they have bloody, mucous stools, tenesmus and abdominal pains among their symptoms; they rarely have fever; there is much less depression, and local examination will show the presence of the cause. A rectal examination should be made in all doubtful cases. This is not an unpleasant task if rubber gloves be used.

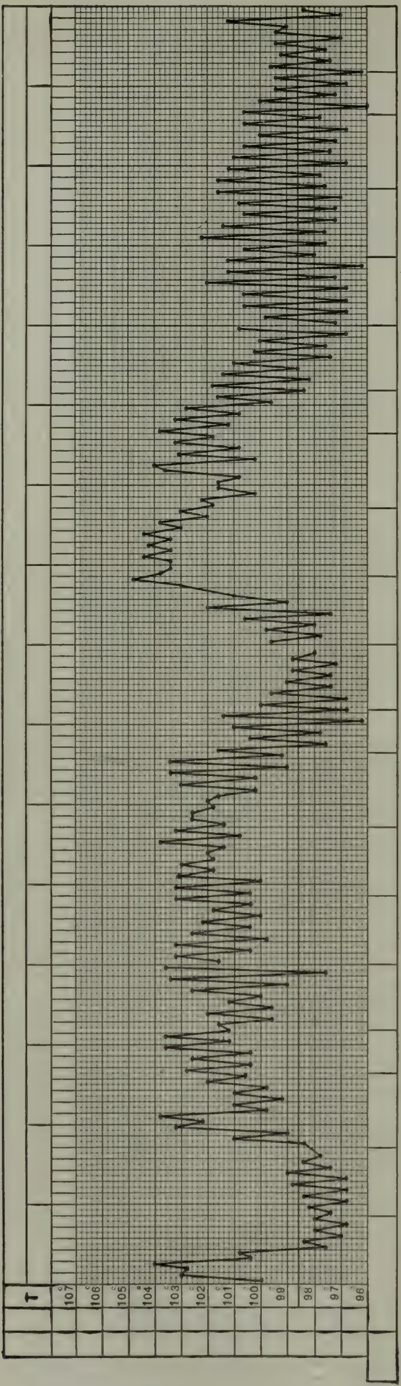

Fig. 11.—Chart of Malta Fever—Case of John H. Musser and Joseph Sailer. Published in Am. J. Med. Sci.

Amebic Dysentery.

Amebic dysentery is a tropical disease. It is usually more chronic in its course than bacillary dysentery. It is prone to have severe colon ulceration as a complication as well as the complication of liver abscess.

The Shiga bacillus can be isolated from the blood in bacillary dysentery; it cannot be in amebic dysentery. Examination of the stools in amebic dysentery will show the *amebæ coli.* They are absent in bacillary dysentery. In certain cases there is an infection by both the bacilli, and the amebae. Here, and in all cases, examination for both organisms must be made. Finally, *amebic dysentery is cured quickly by emetin hydrochlorid, which has no effect on bacillary dysentery.*

12. Malta Fever

Organism.—Malta fever is a protracted, continued fever caused by the Micrococcus melitensis. It is found by Zamit that the fever was spread by the use of goat's milk. From ten to fifteen per cent of the goats of Malta have the Micrococcus in their milk.

Symptoms.—For several days—varying from three to twenty—the individual affected with Malta fever has malaise, headaches, anorexia, constipation and fever.

There is great variation between the morning and night temperature in the accession of the fever. After its beginning, by a "stepladder" increase the fever may reach 103° or 104° F. in the evening; the morning falls amount almost to a remission, and are followed by profuse perspiration (Fig. 11). As the disease progresses the joints enlarge, become tender and painful, and the patient emaciates. At the end of two or three weeks the temperature gradually falls to normal; the patient is then apparently well, but at the end of two or three days of this apparent convalescence, the fever again rises, and he has exactly the same series of symptoms. A series of these attacks of fever, convalescence, and relapses continue for many weeks—often for a year or more. Usually at the end of twelve months the patient is well.

Diseases to be Differentiated from Malta Fever

Malta fever may be mistaken for four conditions:

Tuberculosis
Typhoid fever
Malarial fever
Rheumatic fever.

TUBERCULOSIS.

The diagnosis, however, can be easily made. In the first place—with the exception of tuberculosis—no disease has such a prolonged course. In tuberculosis the regularity of the rises and falls does not obtain that present in Malta fever. Usually also in tuberculosis some local lesion can be found accounting for the prolonged irregular fever.

Examination of the blood will at once make a diagnosis. The Micrococcus melitensis can be cultivated from the blood of Malta fever patients but cannot be cultivated from other conditions. *The Micrococcus melitensis will give the agglutinative reaction with the patient's blood serum, if the case is Malta fever. This will be absent in cases of tuberculosis.*

TYPHOID FEVER.

Typhoid fever has all the characteristics of that disease described in Chapter I. *It gives the Widal reaction for the typhoid bacilli, but does not give the agglutinative reaction with the Micrococcus melitensis.* All of the intestinal symptoms of typhoid fever are absent in Malta fever.

MALARIAL FEVER.

In malarial fever the diagnosis is easily made by finding the organism of malaria which is absent in Malta fever. It is quite possible, of course, for a case of Malta fever to be complicated with malaria—and under these circumstances the presence of the agglutinative reaction for the Micrococcus would make for Malta fever—while the presence in the course of the fever of the malaria organisms will make a diagnosis of malarial fever positive.

Rheumatic Fever.

Rheumatic fever may be much prolonged; joint symptoms are present as they are in Malta fever. However, the joint symptoms in rheumatic fever are much more changeable than they are in Malta fever, affecting first one joint and then another. The fever is also much more irregular in rheumatic fever, and there is leukocytosis. The agglutinative reaction with Micrococcus melitensis is wanting.

13. Asiatic Cholera

Organism.—Asiatic cholera is caused by a spirillum, the comma-bacillus of Koch, cholera vibrio.

Symptoms.—Its chief symptoms are abdominal pains, watery diarrhea, cramps in the calves of the legs, collapse and cyanosis.

Habitat.—The disease occurs usually in epidemics beginning almost without exception in tropical or subtropical regions, spreading frequently with great rapidity to Europe, England and the United States.

Origin.—Epidemics of the disease are all spread by *drinking water* which has become contaminated by discharge from the bowels of individuals suffering from cholera. Any article soiled by such discharge becomes infective—bed linen, clothing, food and drink. Cases have been traced to *contaminated milk* supply, and *vegetables which have been moistened with contaminated water* have distributed the disease. *Flies* have been fouled by discharges, and in turn have contaminated milk and other food. The disease is *not spread directly by the contact of one person with another, but the case infects through the fecal discharges, which in some way have been swallowed.*

Course of Disease.—An ordinary frank case of cholera usually begins with colicky pains and frequent bowel movements; the pain increases and the diarrhea rapidly becomes a furious watery discharge. The discharge is of white serous character and contains flakes of mucus resembling rice floating in water, hence the term—rice water stools. Occasionally there is blood in both vomit and stools, an extremely unusual sign.

Accompanying this stage extremely painful muscle spasms occur, chiefly affecting the calf muscles. Rapidly the case enters the stage of "collapse." The patient loses superficial fat with surprising rapidity; the features become sunken and cyanosis appears. The pulse becomes rapid and feeble; the urine diminishes in quantity, and often a true nephritis occurs. Vomiting which has appeared early in the case persists and becomes distressing and severe; anything taken into the mouth is instantly vomited. Frequently the infection is so severe and exhausting, that the patient dies within twenty-four to forty-eight hours.

After the severe attacks, which do not immediately prove fatal, there is a stage of *reaction* where the patient either slowly recovers, or dies of high fever or of uremia brought about by an existing nephritis.

Often the *temperature* rises to 103° or 105° F. In fatal cases it may reach 109° F. and *after death in these cases it continues to rise.* In certain cases there appears in the later stages an eruption which is in reality an erythema sometimes in the shape of maculae. The patient sometimes sinks into a typhoid state from which he slowly recovers or he may die from inanition.

Cholerine.—In every epidemic there occur scores of cases which have abdominal pain and diarrhea, with little or no disturbance of the general health; to this class of cases is given the name cholerine. The use of this name, cholerine, is *pernicious,* because the disease is a true cholera and can and does disseminate the disease; extremely severe attacks arising from these light cases.

Diagnosis.—During any cholera epidemic all cases of diarrhea should be called cholera until negative findings prove it to be of other origin. The comma-bacillus being positively specific for cholera, it becomes the duty of every practitioner of medicine to see that the stools of every suspected case are examined by a competent bacteriologist, who can by proper tests give a reliable report in twenty-four hours. Awaiting the report, the case should be treated as though it were cholera.

Most governments now have men trained in the art of cholera diagnosis by reason of their familiarity with the examination of stools, both normal and abnormal, to whom specimens can be sent for examination.

Rapid methods now allow a diagnosis of the case within twelve hours of the time of culture.

At the times of invasion of cholera every facility is given to physicians to have their cases examined, and indeed such examinations should be insisted upon by the health boards and carried out by the establishment of rational and practicable laws.

Diseases to be Differentiated from Asiatic Cholera

The conditions with which Asiatic cholera may be confounded are:

Acute enterocolitis (formerly known as cholera morbus, cholera nostra)

Mushroom poisoning (certain cases)

Ptomain poisoning

Corrosive poisons, such as bichlorid of mercury, arsenical or tartar emetic poisoning.

As a rule there is little difficulty in separating these conditions.

Acute Enterocolitis.

Acute enterocolitis offers the chief difficulty. There is not a single symptom of true cholera—not excepting death itself—which does not occur in certain isolated cases of acute enterocolitis. The sudden vomiting, watery diarrhea, collapse, cyanosis, cold sweat, pains in the limbs and fever, often make the cases indistinguishable from those of true cholera. When such a case is isolated and there is no cholera in the country, the rational opinion is that such cases are acute enterocolitis. When cholera exists they are properly considered cholera, until positively proved to the contrary. The only accurate and sure method of diagnosis is *bacteriological examination of the stools,* the tests being made by one skilled in the work, in a well equipped laboratory. This is one of the laboratory proceedings which cannot be done in private practice.

Mushroom and Ptomain Poisoning.

Mushroom and ptomain poisoning both resemble cholera closely, but there is the history of having taken improper food or mushrooms, and the cases are either isolated or in small groups. They never spread, but end with the cessation of the use of the improper food. Examination of the stools will often show in these cases fragments of the offending food, mushrooms, etc., and bacteriological examination will of course be entirely negative for comma-bacilli.

Poisoning by Corrosive Substances.

Poisoning by corrosive substances, such as bichlorid of mercury, gives rise to the same attacks of vomiting and diarrhea, but the stools are bloody and often small instead of being large, watery, and filled with flakes of mucus. There is usually suppression of urine. No cholera bacilli are found in the stools.

Arsenical Poisoning.

Arsenical poisoning also has painful abdominal spasms, diarrhea and vomiting as symptoms, but the history of the cases, occurring only in small groups, makes the case as not likely cholera, while examinations of the stools makes the diagnosis certain. The stools of arsenical poisoning cases do not show vibrones.

14. Plague

Organism.—This is an acute specific fever caused by the plague bacillus—Bacillus pestis bubonicæ.

Origin.—It is proven beyond question that plague in the usual form is spread through the *medium of rats.* The rats become infected with plague; fleas with which the rats are infested become infected with plague bacilli; the flea bites the human being and thus communicates the disease to him. **Pneumonic plague** may be spread through the infected **sputum,** and **bubonic plague** through **discharges from the buboes.**

Course of the Disease.—It is characterized by a short incubation period not lasting more than three or four days. This is followed by sudden fever, depression, enlargement of the lymphatic glands, rapidly fatal pneumonia, or septic or abdominal symptoms. In the bubonic form the glandular enlargement is particularly marked in the region of the groin, though any or all of the glands may be involved.

Various Forms.—The disease occurs in several forms:

Pestis minor
Bubonic form
Primary pneumonic form
Septicemic form
Abdominal form.

In the *mild form*—abortive or ambulant form—the disease is not serious, as the patient is able to overcome the infection. There is slight fever and moderate enlargement of the lymphatic glands, rarely suppuration. When this form is seen, isolated from a known epidemic, the diagnosis is most difficult and demonstration of the plague bacilli is necessary to establish a diagnosis.

In the *other forms* there may be a period of incubation lasting from two to seven days, then a prodromal period in which there is malaise, headache, prostration, nausea, vomiting and vertigo. There may then be a chill, high fever, prostration, extreme exhaustion and all the marks of an extremely severe infection. The accompanying symptoms differ with the particular type of the disease.

The onset is rapid. The fever may come on suddenly and may rise gradually, reaching its maximum in four or five days. It is usually very irregular. In favorable cases it subsides by lysis. In cases which recover, even when they are not complicated, there may be a septic fever due to suppuration of the buboes. In fatal cases the temperature may run high and very suddenly drop to normal, and then rise to be followed by death. Or it may continue very high and death occur in the period of hyperpyrexia. Dull aching pains begin at the seat of the primary bubo. Suppuration frequently occurs in fatal cases. The conjunctiva are reddened; the eyes are sunken; the cornea clouded and the mucous membranes may be hyperemic. The tongue is coated with a white coat, the pharynx is red. The tonsils may be swollen and hyperemic. The skin is often spotted over with subcutaneous hemorrhages; in the first days the skin is hot, but afterwards profuse sweating occurs.

The patient has an anxious expression and rapid respiration; the legs are flexed; the body is flexed, presenting, as some authors say, a pathognomonic attitude. The spleen is enlarged; vomiting is common. The patients are restless; there is delirium—often talking and crying by turns.

In the pneumonic form—that of the terrific epidemic in China in 1910–1911—which caused 50,000 deaths, the symptoms relied upon for diagnosis were examination of the sputum, which showed enormous numbers of bacilli. When a rise of temperature and increased pulse rate occur, a sputum examination of the blood by culture, or microscopically, will usually show the plague bacillus. The absence of marked physical signs in the lungs, absence of labial herpes, nonpurulent sputum and painless cough, are in favor of plague pneumonia, while the presence of piping râles is unfavorable to plague pneumonia.

In septicemic plague the onset is sudden; the fever is high; all of the glands are enlarged and death occurs within 18 to 48 hours. Examination of the blood is necessary for a diagnosis.

The abdominal form of plague has all the symptoms of a severe infection, with vomiting of blood and bloody stools.

Diseases to be Differentiated from Plague

The disease can be confounded with:

Simple febrile adenitis
Tubercular adenitis
Syphilitic adenitis
Pneumonia—due to pneumococcus
Acute septicemia from other organisms than plague bacillus
Acute enteritis.

As above stated, in the time of an epidemic one would be in danger of considering any of the above conditions plague, and in the China epidemic, cases of pneumococcic pneumonia were sent to the detention hospital. On the other hand, cases of plague could in the beginning, before an epidemic is established, be mistaken for any one of the above conditions.

SIMPLE ADENITIS—TUBERCULAR ADENITIS.

Simple adenitis with fever might be mistaken for a mild form of plague, because there are no distinctive characteristics of the latter except the history. Examination of the glands by aspiration in tuberculosis or simple adenitis would show a negative culture for Bacillus pestis, and in tubercular adenitis, the glandular enlargement is apt to be longer in its appearance. In simple adenitis the enlargement may take place very quickly. History of cases of plague in the vicinity will of course put one on his guard. Adenitis due to chancroid will show the local lesion.

SYPHILITIC ADENITIS.

Syphilis may have fever and general adenitis as early symptoms, but there is always a primary sore in some portion of the body and there is likely to be a secondary rash. Plague bacilli are never found; the Wassermann reaction is present.

PNEUMONIA.

Pneumonia could be mistaken for plague, but there is much less general depression—the physical signs are much more likely to be prominent in pneumococcic infections than in plague infections. Plague bacilli are found in abundance in sputum of plague patients, and not found in croupous pneumonia. The mortality is almost 100 per cent in plague; much less in pneumonia.

ORDINARY SEPTICEMIA.

The case is less severe than in plague septicemia; there are less general glandular enlargements. In certain cases there is the history of a wound. Blood cultures will show plague bacillus in the plague form, and other organisms in the ordinary septicemia.

ACUTE ENTERITIS.

Acute enteritis is much less severe; there is seldom blood in the vomitus and feces; there are no plague bacilli in the discharges.

15. Tetanus

Organism.—Tetanus is due to an implantation of the tetanus bacilli.

Origin.—The infection, without exception, takes place by reason of the tetanus bacilli being brought into contact with the body through some traumatism; a fall, a blow, a crush or anything which abrades the surface, at the same time introduces the tetanus bacillus into the wound. Punctured wounds made by infected material, wounds which are contaminated with dirt which has been fouled by horses, and wounds from cheap powder such as blank cartridges, are especially likely to be followed by tetanus.

Cases are unfortunately on record where infected smallpox vaccine matter has caused the disease, after vaccination—though this has occurred on but few occasions. Wounds from blank cartridges are peculiarly apt to give rise to tetanus. The bacillus frequently obtains entrance into the body by means of the umbilical cord (tetanus neonatorum), soiled dressings for the cord being used. It also occurs as a puerperal infection.

Symptom Complex.—Unfortunately the symptom complex which is recognized as the disease tetanus, is almost an end result. The patient, in the words of one writer, is beginning to die when we first see the initial symptoms.

Course of Disease.—The disease begins with the growth of the bacillus in the wound; the toxin is produced at the site of invasion and very soon attacks the nerve trunks in the vicinity of the wound and unites with them. It then rapidly attacks the spinal cord and the symptoms begin to be observed. On this account, *treatment at the time of the reception of the wound is the obvious and rational procedure.*

The wound, as has been said, may be trivial—the writer has seen rapidly fatal tetanus arise from a mere abrasion of the skin of the chin due to a child falling. It may occur from a vaccination wound on which contaminated matter is implanted after vaccination, or it may follow a crush. The two important factors are a wound and tetanus bacilli. As the tetanus bacilli live and thrive in the earth contaminated with horse refuse, any contamination with ordinary earth—especially near stables—is dangerous. After the reception of the injury the wound may rapidly heal or suppuration take place, depending altogether upon whether pus-forming organisms have gained a foothold. This does not seem to make the slightest difference as to the development of tetanus. As a matter of fact, if there is any difference, it consists in the fact that an individual with a wound well healed over is just a little more likely to develop tetanic symptoms than one who has an open wound.

Symptoms.—The first symptom complained of (occurring from the second to the fourteenth day) is often stiffness of the jaw muscles—a mere pain experienced when the jaws are closed. The muscles at the back of the neck stiffen; rapidly the other muscles follow and become painful. The temperature rises; the reflexes are increased; soon general tetanic convulsions occur. The whole body stiffens and remains in a state of rigid spasm for several minutes; the spine becomes affected likewise and opisthotonos results. The jaws are tightly clinched; if it chance that the tongue or cheek gets in between the teeth, it will be severely bitten. The face is forced into a grinlike convulsion; the lips become cyanosed, and respiration impeded. The appearance of the face has always seemed to the writer to be a grin resembling that of merriment. The term, sardonic grin, is more applicable to strychnin poisoning. In a light case when not disturbed, the patient will lie perfectly still and will talk rationally and coherently between the attacks of spasms; but when disturbed, when there is a noise or a light in the room, the patient will often be thrown into a tetanic condition.

When the attack is severe the spasm will be almost continuous, the patient lying rigid as iron. He can be lifted into an upright position by the head. The patient breathes with difficulty, inspiration being apparently impeded by laryngeal spasms. The temperature rapidly rises and death ends the scene.

In chronic tetanus the symptoms come on slowly and are less severe than in the cases of acute onset. There are usually the cases where the time between the reception of the wound and the onset of the symptoms is somewhat prolonged.

Prognosis.—Cases, which are undoubtedly true tetanus, recover, especially under modern treatment. When the time between the reception of the wound and the first symptoms is over one week, the case may get well without treatment. The mind is clear throughout the attack, the pulse rapid.

Diseases to be Differentiated from Tetanus

Tetanus may be mistaken for:

Strychnin poisoning
Tetany
Hysteria
Hydrophobia
Meningitis.

In all of these there is an absence of the necessary wound through which the infection can occur.

STRYCHNIN POISONING.

Strychnin poisoning often has the history of the drug having been taken. There is the *absence* of the stiffening of the jaw and the reflexes are extremely increased even in slight cases. If strychnin doses have been large, death often occurs with the first or second spasm. Examination of the stomach contents shows the presence of strychnin.

TETANY.

Tetany may occur in children who have rickets, and in gastric dilatation, from any cause. It occurs in pregnancy. The thumbs are drawn into the palms, the toes are flexed and the feet extended. An attack may be induced by pressure over the great vessels. There is likely to be laryngismus stridulus. There is no lockjaw.

HYSTERIA.

In hysteria the patient is usually a female. The attacks are sudden. There is no fever. Often the convulsions appear much more severe than in tetanus.

Pressure over the supra-orbital nerve will usually bring a patient to consciousness; a sudden shock of cold water, or a syphon of water sprayed in the face will do the same thing. The attack often follows a fit of anger.

HYDROPHOBIA.

In hydrophobia general spasm or locked jaws is not present. The patient will at first take liquids with avidity, then as the disease progresses the attempt to swallow will be about as follows: the patient grasps the vessel containing the liquid, tosses the water into the mouth with a sudden motion, thus bringing on a sudden and severe respiratory spasm in which he tosses about, fights for breath and tears at his throat with his hands. There is the history of a bite from some animal.

MENINGITIS.

Meningitis has opisthotonos as one of its symptoms with general clonic, not tetanic, convulsions. There is fever, strabismus, unconsciousness, leukocytosis. Spinal puncture will give a fluid containing the infecting organism.

16. Glanders

Organism.—Glanders is an infectious and contagious disease common in horses. It is caused by the Bacillus mallei.

Symptoms.—The disease is characterized by fever, depression, marked local inflammation of the mucous membranes of the nose and by formation of granulomata, and usually by infection of the glands in other portions of the body. As a result of these granulomata breaking down and suppurating, a general septicemia is often caused—called *farcy.*

Habitat.—The disease is primarily found in animals like the horse, and is occasionally transmitted to man.

Various Forms of Glanders.—It takes four different forms:

(1) Acute glanders
(2) Chronic glanders
(3) Acute farcy
(4) Chronic farcy.

(1) ACUTE GLANDERS usually begins with chilly sensations, fever, malaise and aching of the limbs. The fever may be severe and resemble that of malarial fever or of septic infection. A nasal discharge occurs; the nasal mucous membrane is found to be the seat of a granular inflammation which soon ulcerates and discharges bloody pus. If the case progresses, swelling and induration of the hands and feet occur; all of the lymphatics become involved; about the nose and lips the discharge irri-

tates the parts, which soon become swollen and tender. The whole face becomes the seat of a cellulitis. Over this area an eruption occurs which somewhat resembles smallpox, but the rash does not have the regular development so characteristic of smallpox. Instead of ending in crusts, as does the smallpox rash, the skin may become ulcerated or gangrenous.

(2) CHRONIC GLANDERS usually appears late after an attack of infection, where the portal of entry has been in other parts than the nose; it often follows farcy. There are multiple chronic abscesses in various portions of the body, some of them very deep and leading to the surface by sinuses. The lungs become the seat of abscesses as a result of an exacerbation of the disease.

(3) ACUTE FARCY differs from acute glanders practically only in the fact that the disease does not begin in the mucous membrane of the nose. There is an infected wound; the lymphatics become involved; and rapidly a general bacteriemia occurs, the nose becoming involved later.

(4) CHRONIC FARCY is simply slower in its beginning than acute farcy and follows as the result of a poisoned wound. This condition resembles closely a simple infected wound. Multiple abscesses may appear, however, on the legs and arms.

Diseases to be Differentiated from Glanders

The condition may be mistaken for:

Septicemia
Smallpox
Malarial fever
Syphilis
Tuberculosis.

GENERAL SEPTICEMIA.

The diagnosis must always be made by cultivation of the organism from some of the discharge, by injecting a guinea-pig with the pus, or by the presence of a reaction similar to tuberculin reaction where the mallein is used instead of tuberculin. In septicemia, too, a blood culture will show streptococci or other infecting organism. A serum reaction may be used; the blood serum of the infected individual will agglutinate the Bacillus mallei.

SMALLPOX.

The case in the *eruptive stage* might be mistaken for smallpox. However, in smallpox, the rash occurs over the body on exactly the end of the third day. It is first macular, becoming nodular, then vesicular, and finally pustular.

MALARIAL FEVER.

Cases of farcy or glanders might be mistaken for this condition if the temperature were taken as the chief guide, but the blood of the malarial patient shows the plasmodium. Bacillus mallei cannot be cultivated from the blood.

SYPHILIS.

Some cases of glanders with much distention of the nasal tissues or chronic ulcers might be mistaken for syphilis, but the Treponema and not the Bacillus mallei can be found in the discharges. There is a history of local lesion and of secondary rashes of syphilis which are wanting in glanders.

TUBERCULOSIS.

Some of the chronic cases of glanders with long standing ulcerations may be mistaken for tuberculosis, especially if there be lung lesions, but the presence of tubercle bacilli, and the characteristic consolidations in tuberculosis, will make the diagnosis.

17. Anthrax

Habitat.—Anthrax is a disease common in the lower animals, such as cattle, sheep and goats, although it occurs in other mammals.

Origin.—It is due to infection by anthrax bacilli. Infection of human beings is caused by handling infected hides, hair and wool, through bites of insects such as mosquitoes which have become infected by feeding upon animals sick or dead of anthrax; it also occurs through taking infected milk, butter, meat, etc. The disease therefore is most common in individuals whose occuption causes them to handle raw products such as hides, hair and wool.

Local Lesion.—The local lesion of the malignant pustule is quite characteristic. There is great edema, much more than in ordinary carbuncle. There is little or no pus. The center is black and gangrenous, and the periphery is the seat of small pustules or papules.

Diagnosis.—The diagnosis may be made positively by cultivation or staining of bacillus from the exudate. It is suggested by the absence of pus, and of the so-called "core" of the boil or carbuncle.

Various Forms of Anthrax.—MALIGNANT PUSTULE—EXTERNAL ANTHRAX—*Local Symptoms*—Usually this form of the disease begins as a local inflammation. Here there is an itching noticed which first gives rise to a papule; soon a pustule forms which rapidly becomes gangrenous, leaving a gangrenous center and around it an area of small pustules (Fig. 12). There is not much pain, but a great deal of swelling and edema of the surrounding parts occur. According to the degree of general infection, the patient is seriously or slightly ill, though even in the mildest malignant pustule there is considerable general depression.

Additional Symptoms.—The symptoms in addition to the local ones are fever, depression, also diarrhea, delirium, coma, rapid weak pulse and profuse sweats, and occasionally death in forty-eight hours or more. If the case ends favorably, the slough separates and the patient makes a rapid recovery.

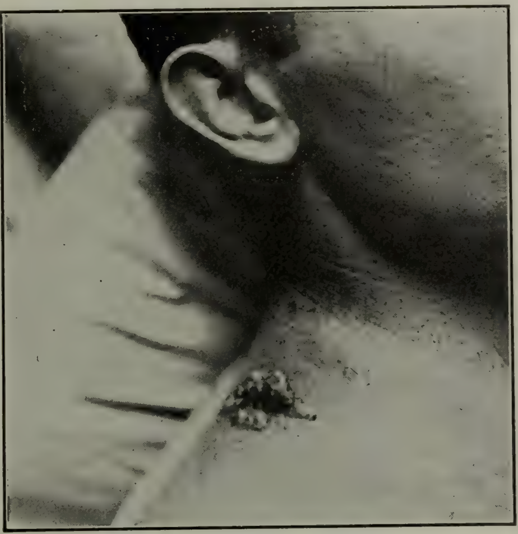

Fig. 12.—Malignant Pustule. (Personal Observation.)

Malignant Anthrax Edema makes its appearance first as a local edema without the appearance of a local papule. The edema very rapidly spreads until the entire part is affected with a tense edema which soon gives way to a gangrenous sloughing area. Anthrax bacilli may be cultivated from the exudate.

Wool-sorters' Disease—General Infection. — The disease is occasionally a general infection from the first. It begins locally in the bronchial mucous membranes—bronchopneumonia, bronchitis, high fever, hemorrhagic expectoration, profuse sweats and collapse may occur. This is perhaps the most dangerous of all of the forms of infection, the patient dying within twenty-four hours with signs of an extremely acute pneumonia. This form is apt to occur in wool houses in which the raw material is cleaned or sorted. It may affect many of the individuals handling a particular lot of wool.

Intestinal Infection begins with the symptoms of a severe intestinal poisoning—sudden fever, chill, nausea, vomiting, diarrhea, vomiting of blood and bloody stools. It is the least frequent form in man—the result of taking infected milk or meat into the system.

Symptoms of Diseases to be Differentiated

Carbuncle—Boil.

The **local inflammation** may be mistaken for a carbuncle, or an ordinary boil or carbuncle or infection by Bacillus ærogenes capsulatus.

Both carbuncles and boils occur in individuals who have not been exposed to contaminated hides or wool. The edema and induration do not extend so rapidly in these conditions, nor is the gangrenous center of the pustule surrounded with papules which is characteristic of anthrax, found in carbuncles or boils. Carbuncles and boils are rarely fatal except in weak individuals. The anthrax bacilli cannot be cultivated from the discharges of either a carbuncle or a boil.

Bacillus ærogenes capsulatus Infection.

Malignant anthrax edema may be mistaken for infection by the Bacillus ærogenes capsulatus, but in the latter there is no history of handling infected animal products, there is a very rapidly developing emphysema of the surrounding parts, and the Bacillus ærogenes capsulatus can be cultivated instead of the anthrax bacilli.

The general infection may be confused with pneumonia, ptomain or corrosive poisoning and plague.

Pneumonia—Ptomain or Corrosive Poisoning.

The **general infection** when it affects the lungs, may be mistaken for pneumonia of great severity. If it affects the mucous membranes of the intestines, it resembles ptomain or corrosive poisoning of the intestinal tract.

The *pneumonic form* can perhaps be recognized by the fact that the usually severe pneumonia is in an individual who is handling hides, hair or wool of animals, which might have been infected with anthrax, and by cultivation of the anthrax bacillus from the sputum. The same may be said of the *intestinal forms* where the anthrax bacillus may be cultivated from the vomitus or from the intestinal discharges.

Plague.

The pneumonic form of plague might be mistaken for anthrax, but the Bacillus pestis will be the infecting organism discovered. Then, too, there is an epidemic of plague. The difficulty might, however, arise where a wool or hide worker is affected with pneumonia during the prevalence of plague.

18. Leprosy

Organism.—Leprosy is a chronic, infectious disease caused by the Bacillus lepræ.

Period of Incubation.—The period of incubation of leprosy is at the present time unsettled.

General Symptoms.—It is characterized by nodules affecting various portions of the skin, by anesthetic areas, by maculae upon the skin, by ulceration of the phalanges, ulceration of the nasopharynx, and involvement of the vocal cords.

There are certain symptoms which may fairly well be called prodromes. Of these, fever of an irregular, intermittent type is perhaps the most characteristic. Then neuralgias, joint pains, malaise, and peculiar attacks of sweating may occur. During the course of a well-developed attack of leprosy, a fever of rather irregular and unusual type may also occur.

Special Symptoms.—Skin.—The first positive manifestations are those of the skin lesions. The disease may attack either the skin or the nerves, either simultaneously or separately.

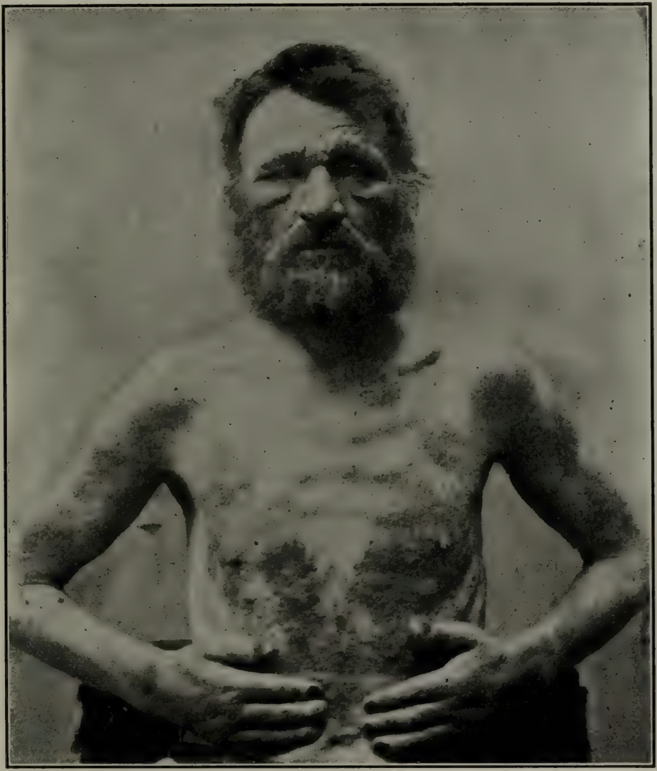

Fig. 13.—Tubercular Leprosy. (Kindness of Dr. Geo. Dock.)

Sometimes when the skin is attacked first there is a rather sudden outbreak on the skin represented by bullae and erythematous patches. The erythematous patches attack the cheeks, the arms, the legs and the buttocks, and do not fade away on pressure. Sometimes they entirely disappear but usually they thicken; nodules appear, and the edges of the patch become raised and nodular.

The skin lesions are always first represented by bullae, which break and leave nodular masses which are symmetrical and bilateral. If the patch affects the face, as it is very likely to do, the face becomes distorted and leonine in appearance (Fig. 13).

Nervous System.—When the nerve is affected, there is thickening in the capsule along the trunk and sometimes nodules on the nerve. At the terminal branches there is hyperasthenia, disappearance of pigment, and often ulceration. This affects the hands and feet especially. The lesions are apt to ulcerate and cause spontaneous amputation of the fingers and toes. Finally when the whole nerve trunk is involved, the area becomes anesthetic. A positive diagnosis can be made by demonstration of lepra bacilli in the skin or discharges.

Diseases to be Differentiated from Leprosy

Leprosy may be mistaken for:

Lupus vulgaris
Syphilis
Erythema multiforme
Multiple Sarcoma
Raynaud's disease
Thrombo-angiophlebitis
Syringomyelia

LUPUS VULGARIS.

Lupus vulgaris affects the face; there is no anesthesia; there is a line of papules along the edge of the patch. It is well affected by x-ray. Examination of the skin excised will show tubercle bacilli instead of lepra bacilli. The differentiation of these bacilli requires the aid of an expert.

SYPHILIS.

Syphilis has a distinct portal of entry represented by the primary sore usually upon the genitals. There are secondary lesions; there may be fever. There is a Wassermann reaction; this sign, however, is not of great importance because a Wassermann reaction occasionally occurs in leprosy. Spirillae may be demonstrated in the lesion. Lepra bacilli can be demonstrated in the leprous lesions.

ERYTHEMA MULTIFORME.

Erythema multiforme shows evanescent lesions. They fade upon pressure; they are not anesthetic and do not contain lepra bacilli. The condition is acute and is accompanied by fever.

MULTIPLE SARCOMA.

Multiple sarcoma do not have the chronic history of leprosy; they are distinct tumors and examination shows spindle or round cells, and no lepra bacilli.

RAYNAUD'S DISEASE.

Raynaud's disease is preceded by intermittent attacks of pallor and cyanosis of the parts; they are not anesthetic and the face is not affected.

THROMBO-ANGIOPHLEBITIS.

Thrombo-angiophlebitis affects Hebrews most frequently. It attacks their great toe. There is no anesthesia, no lepra bacilli in the parts, and examination will show a general arterio venous thrombosis.

SYRINGOMYELIA.

Syringomyelia may show anesthetic areas simulating chiefly true leprosy. Examination of the affected part will not show the lepra bacilli, and leprosy does not have the nervous disturbances, loss of pain and heat sensations of syringomyelia.

19. Tuberculosis

General Statements.—This disease has the same cause whichever the organ affected. It is infectious and contagious, being due to an implantation of the Koch bacillus, the Bacillus tuberculosis (Plate 2). Necessarily the symptoms vary with the organ affected, but almost without exception there is fever, emaciation, loss of appetite and exhaustion.

The subject of general or miliary tuberculosis will be considered, and following this the tuberculosis of the various organs will be given special attention.

(*a*) *General or Miliary Tuberculosis*

Symptoms.—General tuberculosis, miliary tuberculosis, begins as a continued fever. There may be some prodromal symptoms of malaise, weakness and pains over the body, but usually the first indication of serious trouble is a continued fever frequently mistaken for typhoid fever. Indeed, unless there be an initial lesion—which can be diagnosed—of a particular organ affected with tuberculosis, or unless some organ show signs of involvement during the course of the malady, there is great difficulty in making a diagnosis. With the prostration, the pulse is unduly rapid, and there is often frequent respiration. Absence of leukocytosis, great emaciation, restlessness and delirium all characterize this affection.

Duration.—The length of time the disease last varies greatly.

Diagnosis.—If meningitis supervenes or if the lungs become soon affected, as they are likely to do, the case is recognized as one of meningitis, lung disease, or a disease of the organ affected. The abdomen is usually retracted. The only condition with which this condition is likely to be confounded is typhoid fever. There are no characteristic typhoid signs. The characteristic tongue, diarrhea, abdominal distention, rose spots, hemorrhage, etc., of typhoid fever are wanting; Widal reaction is absent. There is leukopenia; there may be no von Pirquet reaction.

When the lungs are affected, or when meningitis or peritonitis supervene or are the starting points of the general infection, the condition of the organ affected is usually recognized, but unfortunately for the accuracy of the prognosis, the general tuberculosis is often overlooked.

(*b*) *Tubercular Adenitis*

Characteristic Features.—This is characterized by glandular enlargement, sometimes a simple enlargement of the gland, frequently progressing into a cheesy and purulent degeneration. This adenitis may affect a single gland, a mass of glands in a particular region, or there may be a very general glandular enlargement affecting practically every gland in the body.

Diagnosis.—To differentiate the various forms of adenitis is difficult and often impossible. When one or more of the glands suppurate and examination shows masses of cheesy material, the case is unquestionably tuberculosis, or if the privilege be given to remove a gland and section it, a positive diagnosis may be made by the presence of tubercle bacilli in the gland.

Conditions to be Differentiated from Tubular Adenitis

This form of tuberculosis may be confused with:

Leukemia
Hodgkin's disease
Syphilis
Irritative adenitis.

Leukemic Adenitis can always be diagnosed by a differential and numeral blood count. Here the increase of leukocytes, the preponderance of lymphocytes, and the presence of myelocytes, eosinophils, and nucleated cells in certain forms are indisputable proof of leukemia. It never occurs in tuberculosis.

Hodgkin's Disease is more difficult to distinguish. Here there is no marked leukocytosis; a persistent slight increase of white cells, increase of the transitional leukocytes and blood platelets favor Hodgkin's disease. The glands most frequently involved in Hodgkin's disease are found just above the clavicle and filling the lower trapezoid sternocleido triangle, with large substernal and bronchial masses.

In cases of doubt, excision of a gland and its microscopic examination will make the diagnosis. Tubercle bacilli are not found in glands of

Hodgkin's disease; the tuberculin reaction is absent in both leukemia and Hodgkin's disease. The presence of *von Pirquet tuberculin reaction* in children under one year of age is good proof of the ADENITIS being TUBERCULAR. When the patient is over one year of age a positive *von Pirquet* reaction simply means a tubercular implantation somewhere.

SYPHILITIC ADENITIS must be differentiated by the history or presence of an initial syphilitic lesion, together with the presence of rash, alopecia and mucous patches, and finally by the prompt disappearance of the adenitis under specific treatment. The *presence of Wassermann reaction* and finally, *demonstration of the spirochete* in an enucleated gland, makes a positive diagnosis of syphilis.

IRRITATIVE ADENITIS due to a local infection such as a suppurative tooth, a wound in the neighborhood, or to a chancroid, can be at once differentiated by the presence of these causes.

The enlargement of glands due to so-called glandular fever is distinguished by the sudden appearance of the glands and their disappearance with the end of the attack. The *von Pirquet test* is of some value—*especially when negative.*

ADENITIS ACCOMPANYING OR FOLLOWING SCARLET FEVER, MEASLES, ETC., can be differentiated by the signs of these various diseases.

When the adenitis affects the mesenteric glands the patient emaciates very rapidly and masses can be felt beneath the retracted abdominal walls. This condition must be differentiated from a new growth other than tuberculosis. Retroperitoneal sarcoma, and hypernephroma and enlarged spleen are the conditions most likely to be mistaken for this form of tubercular adenitis.

In RETROPERITONEAL SARCOMA the mass as a rule is larger, of more rapid growth, the blood is likely to show an increase of the polymorphonuclear elements, while in tuberculosis the lymphoid cells are more likely to be increased.

Both HYPERNEPHROMA AND ENLARGED SPLEEN are unilateral; the masses are less irregular and smoother than in enlarged lymph glands. In enlarged spleen grave blood changes are apt to be present of the leukemia or other types, or there is a history of malaria and a possible presence of malarial organisms in the blood. In enlarged spleen also the notch can be felt almost without exception as a distinct depression along the hard sharp edge.

(*c*) *Tubercular Peritonitis*

Characteristic Features.—Tubercular peritonitis is characterized by a distended or retracted abdomen, irregular fever and more or less pain of a sharp character. Occasionally there is diarrhea, sometimes constipation. If the abdomen is distended the presence of free liquid in the abdominal cavity can usually be diagnosticated by dullness in the flanks and the presence of a wave transmitted from one side of the abdomen to the other when the center line of the abdomen is strongly depressed by the edge of the hand. Occasionally dull areas may be distinguished in various parts of the abdominal cavity.

Infrequently the exudate is not liquid in type but fibrinous, the entire parietal and visceral peritoneum being covered with a thick purulent, more or less fibrinous exudate. Under these conditions there is a retraction of the abdominal walls and there is no liquid in the abdominal cavity, either free or loculated.

Diseases to be Differentiated from Tubercular Peritonitis

The conditions with which tubercular peritonitis may be mistaken are:

Typhoid fever
Chronic enteritis
Appendicitis
Atrophy of the muscle of the intestinal wall (in very young children).

The presence of consolidation of the lung or other evident tubercular lesion always points to tuberculosis as a cause of these abdominal conditions.

TYPHOID FEVER.

In typhoid fever there are rose spots, enlarged spleen, the peculiar tongue, diarrhea and the Widal reaction. Typhoid fever is usually more acute than tubercular peritonitis.

CHRONIC ENTERITIS.

Chronic enteritis, non-tuberculous in type, can be distinguished by the *entire absence* of liquid, or the boardlike hardness of the abdomen when it is retracted. There is less emaciation in cases of enteritis, non-tubercular in type, and the disease is made much worse or is very evidently caused by errors in diet.

APPENDICITIS.

A chronic appendicitis may be so erratic in its course that the unwary may be deceived in thinking that the extensive peritonitis may be the result of a tubercular process. Here the history of the case, the beginning in the right iliac fossa, with local tenderness in that position, with practically always a *polymorphonuclear leukocytosis,* is fairly good proof of the presence of the local inflammatory condition as the cause of the disease. On the other hand enlarged and tender tubercular glands in the region of the appendix may very readily be mistaken for appendicitis. In this condition, however, the leukocytosis is likely to be *lymphatic in type and of* *less severity* than in ordinary appendicitis. Rectal examination will occasionally show a fluctuating mass in appendicitis.

ATROPHY OF THE INTESTINAL WALL.

Atrophy of the intestinal wall, when present, occurs practically always in children who are artificially fed, and there is a uniform general distention of the abdomen without the presence of liquid; frequently the movement of the distended coils of the intestine can be seen through the thin abdominal walls.

(*d*) *Tubercular Meningitis*

Characteristic Features.—The onset of this condition may be sudden or gradual. If the individual be a child, the usual history is several days of malaise, restlessness, irregular fever, anorexia, constipation with gradual loss of weight. After a few days to one or two weeks, the patient evinces symptoms referable to *involvement of the brain*—the previous symptoms being due to a *general tubercular involvement.*

The **brain symptoms** may be ushered in by a severe general convulsion, followed by stupor, strabismus, blindness, stiff neck, clonic or tonic convulsions of the extremities. Kernig's sign, inability to extend the leg on the thigh when the thigh is held at right angles to the trunk, is usually present, as is also Babinski's sign. Tache cerebral and intermittent flushing and paling of the face are common.

The case on the other hand may develop, so far as the brain symptoms are concerned, much more gradually. Headache may be the first symptom. Some paralytic symptom such as strabismus, paralysis of one limb, loss of vision (this paralysis frequently being fleeting in character), stiffness of the neck amounting frequently to opisthotonos, is very frequent.

Course of the Disease.—The child usually has more or less constant convulsions toward the end of life, the eyes become the seat of a serious conjunctivitis. Cheyne-Stokes form of respiration becomes the type of breathing, sometimes appearing soon, sometimes late.

Diagnosis.—Finally spinal puncture may be depended upon. The fluid is almost entirely clear, but upon long centrifugation and staining with carbol fuchsin, tubercle bacilli may be demonstrated. Usually there is an increase in lymphoid cells in a spinal fluid from tubercular meningitis. A röntgenogram will often show enlarged peribronchial glands.

Diseases to be Differentiated from Tubercular Meningitis

The conditions to be distinguished are:

Typhoid fever
Pneumonia with cerebral symptoms
Various other forms of meningitis
Cerebrospinal fever (meningococcic meningitis).

Typhoid Fever.

In the early stages when there is merely an irregular fever, before brain symptoms are prominent, the condition may easily be mistaken for typhoid fever. However, in typhoid fever there is a continued rather than an irregular fever, the enlargement of the spleen, the occurrence of diarrhea, and distention of the abdomen—as opposed to retraction of the abdomen and constipation. The absence of a conjunctivitis, the absence of sudden flushing and paling of the face or extremities, especially when handled, all speak for typhoid fever as opposed to tuberculosis of the meninges. The Widal reaction is present in typhoid fever. There is less emaciation in typhoid fever than in tubercular meningitis.

Spinal puncture will show a clear fluid in both typhoid fever and tubercular meningitis, but tubercle bacilli can be demonstrated in tubercular meningitis after painstaking search.

Pneumonia.

Pneumonia often has among its early symptoms, cerebral conditions which closely simulate meningitis. The *pulse temperature rate is not apt* to be *disturbed in meningitis;* the *respirations are rapid in pneumonia.*

In pneumonia, even in the early stages, there are certain physical signs which mark the case—dullness together with rather faint and distant voice sounds being in the experience of the writer the earliest signs of pneumonia, especially in children in whom the differential diagnosis between pneumonia and meningitis is likely to be involved. Examination of the spinal fluid will show the absence of tubercle bacilli.

Cerebrospinal Fever.

Meningococcic meningitis begins suddenly. There is usually herpes, anesthesia of the conjunctiva, intense headache, early Kernig and Babinski signs together with rapidly appearing stiffness of the neck. Occasionally there are petechial spots. Almost without exception there is a high polymophonuclear leukocytic blood count, rising often to 20,000 leukocytes to the cubic millimeter. In tubercular meningitis there is not apt to be a high leukocyte count.

The absolute differential diagnosis is obtained by spinal puncture. In tubercular meningitis, as stated before, there is almost without exception a perfectly clear spinal fluid showing on centrifugation, tubercle bacilli and a very few leukocytes, largely lymphocytes. The fluid in epidemic cerebrospinal meningitis, however, is apt to be cloudy, and this cloudiness is due to many polymorphonuclear cells. Within these cells are found meningococci. Other forms of meningitis due to pneumococci, streptococci, etc., are also distinguished by their spinal fluids. Almost without exception in these latter infections the fluid is turbid with polymorphonuclear cells, but the staining of the fluid will show the infecting organism in the individual case. The organism is usually outside the cell and rarely within it.

(*e*) *Tuberculosis of the Lungs*

Site.—The usual site of implantation of tuberculosis of the lungs is at the *apex* of one or the other lung. It is true that other portions of the lung may be first affected and *great care must be taken that all portions of the lungs are examined.*

Characteristic Features. — The symptoms of lung involvement by tuberculosis are often insidious. Occasionally they are abrupt and take on the form of an ordinary croupous pneumonia. The usual picture is as follows: slight morning dry cough, indisposition to attend to the usual duties of life, failing appetite, slight but progressive anemia, slight but continuous loss of weight. Gradually the cough increases; expectoration appears and increases; indisposition increases to distinct illness; slight anemia becomes grave; emaciation becomes evident; extreme diarrhea appears alternating with constipation.

Occasionally the first symptoms to attract attention of the patient or his friends is a *pulmonary hemorrhage.* Careful questioning often fails to establish a previous history which deviates in the least from that of perfect health.

Again the onset may be sudden in the form of a *very rapidly developing croupous pneumonia.* Frequently in this form the expectoration is markedly bloody, often amounting to a true pulmonary hemorrhage. The fever, however, is likely to continue beyond the time for its disappearance in true pneumonia, and characteristic of the disease, is likely to be prolonged. Frequently these supposed cases of croupous pneumonia due to pneumococcic infection have their origin, however, in or around an old tubercular lesion unknown or unsuspected by either patient or physician.

Physical Signs.—In the insidious cases this valuable diagnostic help is extremely difficult to detect early. Examination of the patient should be painstaking, with all the clothing removed from the chest; it should be made with the patient in a good light, and both while he is lying and sitting. A stethoscope must always be used. Inspection is likely to show limitation of motion in a certain portion of the chest, most frequently at one or the other apex, though every single part of the chest must be carefully examined. It is a most pernicious habit to confine one's examination to the apices alone, because many cases have the first implantation in another portion of the chest. Over the area suspected the breath sounds are abnormal: there is either the "granular" breathing or the sound characterized by undulations of both inspiratory and expiratory sounds with a sort of rumbling noise. After coughing and full inspiration, just at the end of inspiration a few distinct râles may frequently be heard, either moist or dry in character.

The expiration may be distinctly prolonged and of somewhat bronchial character. As the case advances these sounds begin to take on the character of distinct blowing breathing, both inspiration and expiration being distinct, the expiration being quite as long as the inspiration even upon quiet breathing. Forced breathing will frequently bring out the characteristic breath sounds when quiet respiration fails. The suspected spot must always be compared with exactly the same spot in the opposite lung.

Tactile fremitus may be either diminished or increased, depending upon the amount of overlying pleuritic involvement. Vocal resonance is likely to be increased, though this too may be diminished for the same reasons as change in the tactile fremitus. The whispered voice sounds will frequently give more difference than the spoken voice sounds.

Percussion will produce a higher pitched note over the suspected area, very frequently amounting to positive dullness. Here the percussion must be made the greatest possible lightness of stroke; a hard percussion will often fail to bring about the slightest difference in note. It must be remembered that all the physical signs are increased in health at the right apex, but the breath sounds in health, while they may be louder at the right than at the left, are not changed in character. Narrowing of the area of resonance on the affected side above the clavicle is a valuable sign.

Sputum Examination.—This is of the utmost importance. Many cases of tuberculosis in the realms of uncertainty are at once made positive by a careful examination of the sputum, which can be done by the general practicing physician. If a multitude of patients or an insufficient training prevent the practicing physician from doing this most important work, then it should not be neglected but should be given into the hands of a competent assistant or should be done at one of the clinical laboratories. Inability on the part of the patient to pay for this laboratory work is not sufficient reason for failure to have it performed. It is claimed by some that tubercle bacilli is a late sign, and a diagnosis should always be made on symptoms and physical signs. In the writer's rather extended experience *physical examination often fails to make certain a diagnosis when sputum examination will decide it.*

Gay and Claypole have lately pointed out the close relationship between *certain forms of streptothrix and tubercle bacilli* from their tinctorial qualities. Care must be taken to differentiate between the two organisms.

Temperature Records.—Careful temperature records taken in the morning and in the evening will usually show in incipient tuberculosis a slight evening rise of temperature.

Blood Examination.—Examination of the blood will almost invariably show a reduced percentage of hemoglobin, with a slight reduction in the red

PLATE II

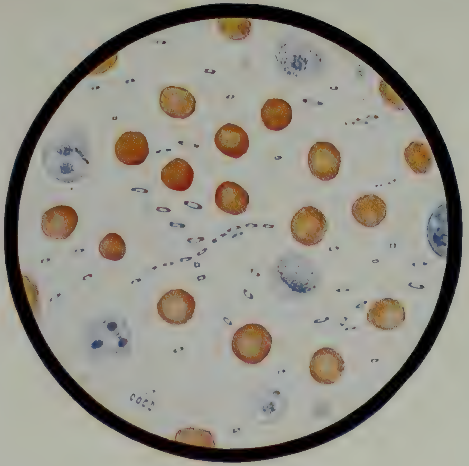

Fig. 1.

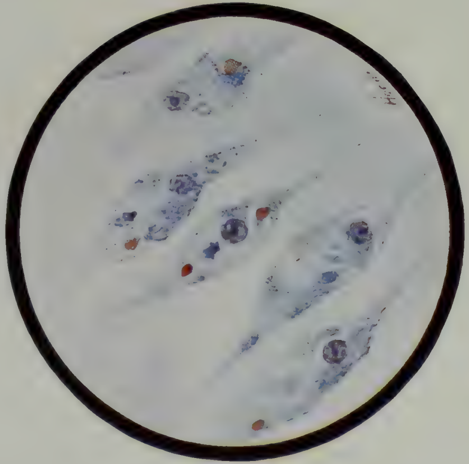

Fig. 2.

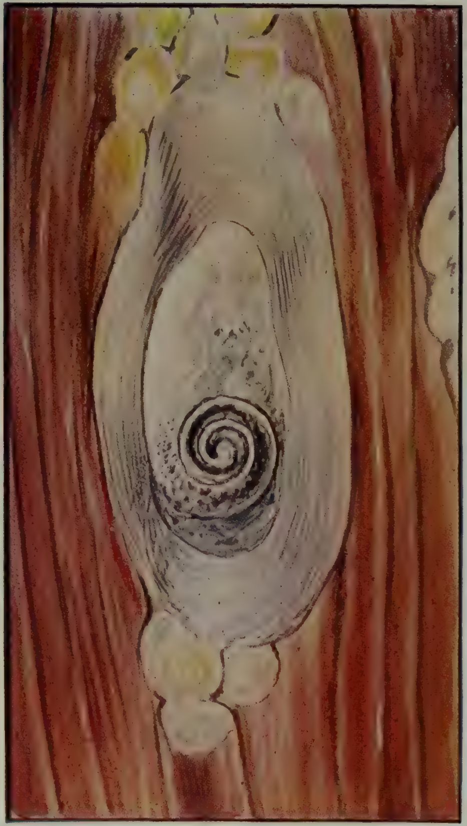

Fig. 3.

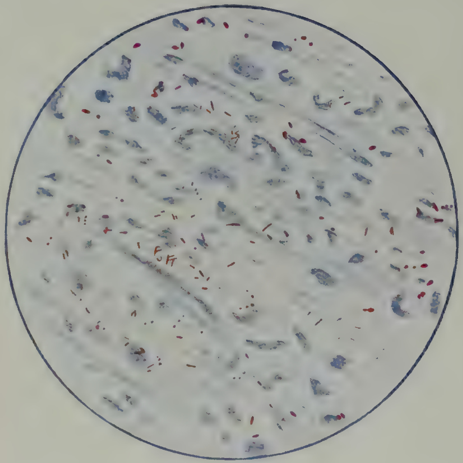

Fig. 4.

Fig. 1.—Plague Bacilli. (Adapted from Wherry.)
Fig. 2.—Negri Bodies in Ganglion Cells. (Univ. of Pa., Vet. Hosp.)
Fig. 3.—Trichinella spiralis. Parasite in Its Cyst, Cut in Section. (After Tyson's "Practice of Medicine.")
Fig. 4.—Smear of Sputum Showing Tubercle Bacilli. (Personal Observation.)

cells. The *absence of a polymorphonuclear leukocytosis will serve to distinguish the condition from a low grade suppurative process.*

X-ray Examination.—X-ray examination with a fluoroscope, or with the plate, will frequently show a shadow on the infiltrated area. The writer believes that a physician should familiarize himself with the readings of radiographs in order that he may himself interpret the plates and not leave this to the radiographer alone.

Diagnosis.—A slight anemia, a constitution in which fatigue is easily produced, failing appetite, emaciation, a slight and persistent evening rise in temperature with or without cough, make a grave suspicion of tuberculosis. Added to these symptoms, physical signs above described, together with a blood which does not show a leukocytosis and shadows in the lungs shown by the x-ray, make a diagnosis practically positive. A history of remote exposure to tuberculosis infection is important.

Tubercle bacilli in the sputum make the diagnosis absolute. Actual pulmonary hemorrhage means, in at least 95 *per cent of the cases, beginning tuberculosis of the lungs, whether the hemorrhage be great or small.*

Conditions to be Differentiated from Early Stage of Tuberculosis

The conditions with which beginning tuberculosis of the lungs can be confounded are many:

Typhoid fever
Malta fever
Marked suppurative process
Malarial fever
Neurasthenia
Chlorosis
Bronchitis
Pleurisy
Hodgkin's disease
Exophthalmic goiter
Syphilis
Hemoptysis
Malignant disease
Mycosis
Heart disease
Aneurism of the arch of the aorta.

TYPHOID FEVER.

Here there is the same difficulty in the beginning as there is in differentiating general miliary tuberculosis. There is fever as an early symptom but the fever is continued in type and not irregular, as in tuberculosis; there is more intestinal disturbance in typhoid fever; there are rose spots which are not found in tuberculosis; an enlarged spleen and Widal reaction are present.

MALTA FEVER.

Malta fever is a continued fever of irregular type due to the infection of the Bacillus melitensis. Here there is entire absence of any of the physical signs characteristic of tuberculosis; there is no sputum containing tubercle bacilli; there is an agglutination reaction with Bacillus melitensis which is not present in tuberculosis. (See Fig. 11 for temperature chart of Malta fever.)

MARKED SUPPURATIVE PROCESS.

A latent SEPTIC PROCESS has irregular fever, but the physical signs are not the same. If the condition, however, is an empyema there may be some cause for doubt. The physical signs in empyema, however, are dullness, dislocation of organs, lack of tactile fremitus—which are the antithesis of those of consolidation.

The blood shows a *leukocytosis,* and not a *leukopenia.* Careful examination in septic processes will usually discover some area of suppuration—appendix, gall-bladder, carious teeth, sinuses, etc.

MALARIAL FEVER.

Malarial fever can always be diagnosticated by the presence of Plasmodium malariæ in the blood. If this for any reason is not practicable, the administration of quinin should be used as a diagnostic measure. If the case is malaria it will disappear with quinin administration. Any fever which resists proper quinin administration is not malarial.

NEURASTHENIA.

Many individuals with incipient, even advanced cases of tuberculosis are neurasthenic. The danger is that the lung condition will be overlooked. Every case of neurasthenia should be carefully and repeatedly examined to exclude tuberculosis.

CHLOROSIS.

Chlorosis is characterized by great reduction of the hemoglobin and moderate reduction of the red cells. It occurs in young girls. Here great care must be taken that a careful measurement of the temperature, chest examination, and a sputum examination be made. Chlorosis has only the blood change, dyspnea and cardiac palpitation as symptoms; rise of temperature, chest dullness or prolonged expiration must be looked upon as extremely suspicious of some other condition.

BRONCHITIS.

Bronchitis is not associated with dullness, blowing breathing, change of breath sounds, fever or emaciation. To continually call a case which has fever, loss of weight, anemia and much sputum "bronchitis" is to deny the patient the chance of being cured.

PLEURISY.

Pleurisy is such a constant accompaniment of tuberculosis, that *every case of persistent pleurisy must be looked upon as probably tuberculous in origin, and for safety's sake, so treated.*

HODGKIN'S DISEASE.

Irregular fever is one of the symptoms of Hodgkin's disease, but the *glandular enlargements, progressive anemia, slight leukocytosis,* and above all the **examination of the blood** will make the diagnosis.

EXOPHTHALMIC GOITER.

Exophthalmic goiter is often mistaken for tuberculosis. Exophthalmos, enlarged thyroid, tremor and tachycardia may be present in some cases of tuberculosis, but it is extremely rare for all of the signs to be present except in true Graves's disease. Careful examination with discovery of an area of consolidation will help to make the diagnosis, and some cases can only be diagnosed by the absence of tuberculin reaction.

SYPHILIS.

Syphilis may, when it affects the lung, have the same physical signs as does tuberculosis, but it is extremely rare. There is the history of syphilis; the Wassermann reaction is present; it responds to antisyphilitic medication. .

HEMOPTYSIS.

Hemoptysis due to cardiac disease may be mistaken for that present in tuberculosis. The careful examination of the heart, the presence of a valvular defect, especially mitral stenosis, and the absence of tubercular lesions, will make the diagnosis. If the case is complicated with infarcts of the lung, it may be most difficult to come to a decided opinion, but the absence of the actual lung condition of tuberculosis will be most valuable.

The later stages of tuberculosis of the lungs can invariably be distinguished from non-pulmonary conditions, by the physical signs in the lungs which have been detailed above, but there may be some *difficulty in deciding whether the lung condition is a consolidation or a collection of fluid, whether if solid the consolidation is malignant, mycotic, or tubercular.*

MALIGNANT DISEASE OF THE LUNG.

Malignant disease of the lung can frequently be confused with tuberculosis. There is cough, but as a rule the cough is free from any large amount of expectoration. The *consolidated area* in malignant disease is found anywhere over the lung area, while in tuberculosis the *site of election* is at the apex. The physical signs over the malignant area are not as plain and distinct as over the consolidated tubercular area, thus breath sounds are apt to be indistinct instead of blowing, fremitus is likely to be decreased rather than increased, as are the voice sounds.

Puncture with a needle over the affected area will prove the condition not to be one of a collection of fluid, as might be suspected from these signs.

Then careful search will likely show some *seat of primary malignant disease,* when of course the likelihood of tuberculosis of the lung is less. The presence of a malignant growth near the heart or other viscera, usually pushes the organ away from its usual position, showing surely that either a liquid or a new growth is present. Indeed, there is more difficulty in differentiating a malignant area from an area with a collection of fluid, than there is a malignant area from one of tuberculosis. What sputum there is in a malignant case, does not contain tubercle bacilli.

PLEURAL EFFUSION.

A pleural collection of fluid will be suspected from the history of the case. An acute tubercular consolidation usually gives such evident signs of dullness, increased fremitus and vocal resonance with râles, that it is at once differentiated as a consolidation and not as a collection of fluid, and usually the history of a lung lesion, supposed to be a collection of fluid, is that of a comparatively acute lesion. *If a serious doubt exists as to whether the lesion is a consolidation or a liquid, the use of an exploring needle is not only justifiable but imperative.* True, a needle does not always find the liquid which exists. An x-ray picture will frequently, but not always, enable a diagnosis to be made. If there is still reasonable doubt as to the presence of fluid, then a detailed exploration should be done to settle the vexing question.

MYCOSIS OF THE LUNG.

A mycosis may simulate the dullness, fremitus, and blowing breathing of tuberculosis—still more the physical signs of an effusion. When the area breaks down and gives rise to fluid, the diagnosis can be made by finding mycotic elements in the liquid, or the organism can be found in the sputum if there is expectoration.

CROUPOUS PNEUMONIA.

Croupous pneumonia might be mistaken for acute tuberculosis of the lungs, but the *short course of the disease and the characteristic crisis* make the case one of pneumonia. If the case is prolonged, however, there will be great doubt as to whether it is one of tuberculosis. The characteristic symptoms detailed above will make the diagnosis.

ANEURISM OF THE ARCH OF THE AORTA.

Aneurism of the arch of the aorta, or a huge heart, has been mistaken for tuberculosis of the lungs, because the compression produced by the aneurismal sac resembles the consolidation due to tuberculous infiltration. An x-ray photograph, however, should settle the question.

(f) *Tuberculosis of the Kidneys*

Incipient Stage.—Tuberculosis of the kidneys ranges from one or more small tubercles embedded in the stroma or pelvis of the kidney, to the condition where the entire kidney substance is changed into a huge tubercular mass. This variation in the pathology gives rise to a great variety of symptoms.

Symptoms.—The smallest degree of infection which gives rise to symptoms is characterized by frequent urination, bloody urine, with none, or only a few, tube casts. If the urine is carefully sedimented, usually tubercle bacilli may be demonstrated in the urine. The presence of acid-fast bacilli, while suspiciously symptomatic of tuberculosis, does not make a positive diagnosis; these organisms may be smegma bacilli. However, the guinea-pig test can be relied upon for positive differentiation. Sometimes there is pain in the region of the kidney or along the line of the ureter, as the lesion advances in degree both leukocytes and blood appear in the urine with numerous tube casts. In the later stages when the kidney is converted into a tubercular sac the urine is highly purulent. In all these stages tubercle bacilli may be present in the urine. Pain is paroxysmal or may be almost constant. The kidney may not be enlarged and may not be tender; on the other hand both enlargement and tenderness may exist. The patient fails in health, becomes emaciated, and finally dies either of simple exhaustion or of general miliary tuberculosis.

Diagnosis.—After the acid-fast bacilli found in the urine are definitely decided to be tubercle bacilli, it must be ascertained by ureteral catheterization that the pus comes from the kidney. It is only in this way one can be sure of the diagnosis. In every case of suspected tuberculosis of the kidney the **cystoscope** and the **ureteral catheter** must be used.

Conditions to be Differentiated from Tuberculosis of the Kidney

CHRONIC NEPHRITIS—PYONEPHRITIS NON-TUBERCULOUS.

This condition is constantly mistaken for chronic nephritis—either of the interstitial or parenchymatous type, and for pyonephritis either of the infectious or calculus type.

The characteristic symptoms of nephritis are as follows:

INTERSTITIAL NEPHRITIS—PARENCHYMATOUS NEPHRITIS.

From interstitial or parenchymatous nephritis the condition may be distinguished largely through the condition of the urine. The urine of tuberculosis of the kidney is purulent in character, or in the very early stages there is hematuria.

The *urine of nephritis* is non-purulent, the main abnormal characteristics being albumin and tube casts. In the interstitial form the specific gravity is continuously low and the color usually light, the tube casts often few in number and of the pale granular type. In the parenchymatous type the urine is large in amount and tube casts are pale granular, dark granular and hyaline. If there is an amyloid kidney there may be waxy casts and a large amount of albumin. In none of the forms of nephritis is there a pyuria. *None of the forms contain tubercle bacilli in the urine.*

In parenchymatous nephritis there is apt to be a large amount of *edema,* which is wanting in tuberculosis.

In the interstitial form of nephritis there is constantly *high blood pressure,* the systolic pressure often reaching 220 mm. of mercury. This is not so marked in the parenchymatous form, and is exactly contrary to the condition of the blood pressure in tuberculosis.

Retinitis does not occur in tuberculosis of the kidney, but is common in interstitial nephritis.

Non-tuberculous pyonephritis has all of the symptoms of tuberculosis of the kidney, with the exception of the presence of tubercle bacilli in the urine.

The cystoscope and urethral catheter will determine positively whether the pyuria has its rise in the kidney and will demonstrate which kidney is affected, and the x-ray will determine whether the suppuration is due to the presence of a renal calculus. Examinations of the urine for acid-fast bacilli will show the absence of tubercle bacilli.

CYSTITIS.

Pyuria and dysuria are symptoms of cystitis. Examinations of the urine will decide whether the latter contains tubercle bacilli, but examinations by a cystoscope are necessary to establish the fact that the pus does not come from the kidney.

(*g*) *Tuberculosis of the Bladder*

Symptoms.—There is hemorrhage, tenderness and pyuria.

Diagnosis.—The local lesion can be distinguished by cystoscopic examination, which shows a more or less raised, red, ulcerated area in the bladder wall. This method of examination will show the difference between such a tuberculous ulcer, vesical calculus, and a malignant growth, with which tuberculosis of the bladder may be confounded. There is no other method known to the writer which makes a diagnosis positive except the presence of tubercle bacilli in the urine as confirmed by the guinea-pig test, and *this will not exclude the kidneys as a focus.*

(*h*) *Tuberculosis of the Testes*

Morbid Anatomy.—Tuberculosis of the testes is characterized by enlargement of the whole organ, which may be quite gradual. It is not overpainful and is usually nodular in type. Occasionally the organ takes on a sudden inflammatory condition, and enlarges rapidly. Puncture of the sac under these conditions shows usually the presence of a purulent material in the growth. If a free incision be made the material will be found to be cheesy in consistency.

Conditions to be Differentiated from Tuberculosis of the Testes

Tuberculosis of the testicle may be mistaken for:

Acute epididymitis
Malignant disease
Gumma.

ACUTE EPIDIDYMITIS.

Acute epididymitis has a sudden onset, is usually a sequel to gonorrheal infection, and is extremely painful. The epididymis is alone involved.

MALIGNANT DISEASE.

Malignant disease is stony hard, has a tendency to ulcerate through the skin, when the growth becomes cauliflower in type. Puncture does not show any liquid.

GUMMA.

Gumma is hard, painless, and responds to antisyphilitic treatment; the individual will give the Wassermann reaction.

(*i*) *Tubercular Enteritis*

Etiology.—Tubercular enteritis rarely exists as a primary infection; as a rule it is secondary to a lung or glandular infection.

Symptoms—Physical Signs.—Its symptoms are pain in the abdomen, diarrhea which is apt to be lienteric in character, with tenderness in varying portions of the abdomen, depending upon the area of the gut which contains the inflamed Peyer's patch or solitary gland. The most usual tender area is the right iliac fossa, because the tubercular patches are more common in the intestine lying in that position. If the solitary glands are much involved, the tenderness is general; fever of rather regular type is a constant symptom; emaciation is often extreme. If the ulceration is far down in the sigmoid, there are signs of tenderness, small painful stools, often with blood and mucus. The abdomen may be much distended, causing great resemblance to typhoid fever; emaciation becomes extreme.

Course of the Disease.—Frequently there are remissions or almost entire subsidence of the symptoms, so that the case seems entirely well. After a lapse of a shorter or longer time, there is a renewal of the symptoms with all the attendant distress. In this manner the case may drag on for weeks or even months, until the patient is exhausted and death ensues.

Conditions to be Differentiated from Tubercular Enteritis

The conditions for which it might be mistaken are:

Simple enteritis
Dysentery
Chronic appendicitis
Typhoid fever.

SIMPLE ENTERITIS.

Simple enteritis is acute in character; it can usually be traced to some definite cause. There is less emaciation, very little if any fever. *Tubercle bacilli* may frequently be found in the stools of tuberculous patients; they are *absent in simple enteritis.*

DYSENTERY.

Dysentery due to tuberculosis of the colon, ordinary bacillary dysentery due to some strain of dysentery bacilli and to amebae, may be confounded.

The acute or specific dysenteries may readily be differentiated by the *acuteness* of their course, and the *presence of the dysentery bacilli.*

The amebic dysenteries of the warmer climates may practically always be distinguished from tuberculosis, by the *absence of tuberculous* *lesions in other portions of the body,* and the *presence of amebae in the stools* of that form of dysentery.

Examination of the stools is not unpleasant and can easily be done in the routine work of the practicing physician.

CHRONIC APPENDICITIS.

Certain cases of appendicitis which have lasted over a long period have disturbance of digestion with frequent stools as symptoms. This with the emaciation which occurs at times leads the physician to make a diagnosis of tuberculosis, but in all such cases signs of inflammation, tenderness, resistance and occasionally a mass in the right iliac fossa, together with paroxysms of pain accompanied with leukocytosis, will prove the case to be one of *local inflammation* and not a general infection.

TYPHOID FEVER.

Occasionally acute tubercular enteritis is mistaken for typhoid fever, but a blood culture or a Widal reaction can be demonstrated in typhoid cases. The diarrhea and recurrent fever which sometimes occur at the end of a course of typhoid fever are very confusing, but dependence may be placed upon the blood examination and upon the fact that no tubercular lesions can be found in any other portions of the body. Tubercle bacilli are absent from the stools of typhoid fever.

(*j*) *Tuberculosis of the Stomach*

Etiology.—Tuberculosis of the stomach exists. It is rare. It is always secondary to grave tuberculosis of other organs, and has to be differentiated from simple ulcer. The history of ulcer of the stomach and duodenum will be seen in another portion of this work.

Symptoms.—The tuberculosis of the stomach gives rise to simple distress rather than the well-marked signs of peptic ulcer.

Diagnosis.—*Diagnosis of tuberculosis of the stomach is practically impossible during life.*

(*k*) *Tubercular Laryngitis*

Etiology.—Tubercular laryngitis is rarely if ever primary. Almost always search will show a lesion in the glandular system, or still later in the lungs.

Symptoms.—It varies from a mere redness or swelling on both of the arytenoids to destructive ulceration, by reason of which the patient becomes entirely invalided and the organ entirely beyond the possibility of functioning. The first symptoms are hoarseness of the voice and a dry unproductive cough—constant and annoying. As ulceration progresses, the larynx becomes extremely painful, particularly upon swallowing. One characteristic of this is *extreme pain in the ear on the affected side.*

The **laryngoscopic picture** is redness, usually beginning in the arytenoids, swelling of these bodies, swelling and redness of the epiglottis as well as of the cords themselves. This soon ulcerates, when an area of ulceration can be found often in the aryepiglottic fold.

Diagnosis.—Tuberculosis can be suspected by a tuberculous lesion in another portion of the body, though *tuberculosis of the lungs or other primary seat of tuberculosis does not of necessity prove the laryngeal lesion tuberculous.*

The *presence of tubercle bacilli* in the sputum, or in the exudate from the larynx, taken directly from that organ, of course proves conclusively the nature of the lesion.

Conditions to be Differentiated from Tubercular Laryngitis

It is to be differentiated from:

Syphilis of the larynx
Simple laryngitis
Carcinoma or epithelioma.

SYPHILIS OF THE LARYNX.

Syphilis can be diagnosticated by the absence of tuberculosis lesions and the known history of the syphilitic infection and also by a negative tuberculin reaction. The presence of the *Wassermann reaction* and the presence of *spirochetes* in the exudate of the larynx is positive of the condition. Syphilis is indicated by the tendency of the lesion to cicatrize, and lastly, by the treatment by mercury or neosalvarsan in clearing up the lesion.

SIMPLE LARYNGITIS.

Simple laryngitis can be distinguished by the absence of tuberculous lésions, the absence of tuberculin reaction, of tubercle bacilli, and the tendency to entire and rapid recovery under mild stimulating local applications.

CARCINOMA—EPITHELIOMA.

A malignant growth does not tend to ulcerate. There is usually a distinct tumor, and also infiltration of surrounding tissues by the new growth. A microscopic examination of the new growth will show the presence of the *characteristic cells* of the carcinoma or epithelioma.

(*l*) *Tuberculosis of the Mouth*

Tubercular ulceration of the buccal surface and of the tongue are rare occurrences.

Conditions to be Differentiated from Tuberculosis of the Mouth

Syphilitic ulcerations
Leukoplakia
Aphthous ulcers.

Syphilitic Ulcerations.

The history of the case is of great value. If the case is one of an evident tuberculous nature, as indicated by lesions in the lungs or other portions of the body and the ulcer is punched out uniform with indurated edges, tuberculosis is the probable cause. If on the other hand there is a history of syphilis, the likelihood of the case being syphilitic comes to the foreground. Here the presence of spirochetae in the scrapings of the ulcer and the presence of a Wassermann reaction will make the diagnosis positive for syphilis.

Leukoplakia.

Leukoplakia may occur as a late syphilitic lesion or as the result of smoking. There is a flat whitish patch; there is no ulceration.

Aphthous Ulcers.

Simple aphthous ulcers are small, covered with a thin white exudate, and extremely painful. They are almost instantly cured by the application of nitrate of silver on the ulcer. No specific organisms are found.

(*m*) *Tuberculosis of the Esophagus*

Esophageal tuberculosis exists. There is no way to exclude the possibility of tuberculosis except by the absence of a general tuberculosis.

(*n*) *Tuberculosis of the Tonsils*

Etiology.—Wood has shown that a certain percentage of cases of chronic hypertrophic tonsillitis have tuberculosis as the causative agent.

Diagnosis.—The differential diagnosis from interstitial enlargement and suppuration in the deep tissues is difficult. Very occasionally tubercle bacilli can be demonstrated in the follicles. Removal and complete dissection of the tonsil will show the true character of the lesion.

(*o*) *Tuberculosis of the Bones*

Tuberculosis of the bones causes a painful, tender, usually enlarged roughened area in the shaft of head of the bone. It is frequently taken for rheumatism and great destruction of the bone is allowed to occur before a proper diagnosis is made and treatment instituted. In the shaft of the bone the symptoms and signs above stated mean one of the following:

Conditions to be Differentiated from Tuberculosis of the Bones

Malignancy
Traumatism
Cysts
Syphilis.

MALIGNANCY.

A malignant condition, sarcoma and mycosis have much the same symptoms as tuberculosis but with less fever, and the case is less chronic. An x-ray will show that the structural tissue of the bone is involved, while in tuberculosis the condition is usually a periostitis with later involvement of the osseous tissue. There is no fluctuation over the area of malignancy, while there is apt to be that condition over a tuberculous area.

TRAUMATISM.

A traumatism always gives a history of an accident. Great care must be taken in eliciting this history that too much stress is not laid upon it, for frequently when a limb begins to be affected with tuberculosis the patient will have a vague remembrance of an injury sustained. Great care must be taken that this injury be not considered the *disease.* It may be the *cause,* but there is now engrafted upon it the disease of malignancy or tuberculosis.

CYSTS.

A cyst at the end of the bone will cause enlargement over the area of disease; it is not painful. The x-ray will show a lack of substance over the area of the center of the cyst.

SYPHILIS.

Syphilis is less easy to differentiate. Syphilis, however, does not have any tuberculin reaction and does give a Wassermann reaction. When the skin breaks down, syphilis does not show areas of suppuration, but shows ulceration instead. Syphilitic lesions are *rarely painful,* as are the lesions due to tuberculosis.

(*p*) *Tuberculous Pleurisy*

General Statements.—Some writers declare that all pleuritis which is not traumatic and which is non-malignant is tuberculous.

Practically all cases of tuberculosis are accompanied by a pleuritis. This form is usually fibrinous or semifibrinous in type and results in a close union of the parietal and visceral pleura which frequently gives no physical signs.

Primary tuberculosis may eventuate in a thick leathery membrane which covers the lung and in fact encloses it in a baglike membrane to which the lung is tightly adherent and from which it cannot be separated. If there is a collection of liquid in the cavity, the liquid is likely to be bloody or purulent, though this is not a necessary characteristic. It may be a serous collection.

Diagnosis.—Occasionally the diagnosis can be made of a pleurisy with or without a pleural effusion. The opinion as to whether it is tuberculous or not must depend upon the seat of an undoubted tuberculous lesion somewhere else and upon the character of the fluid withdrawn. The fluid is likely to contain an excess of lymphocytes; if it contains tubercle bacilli, which is unlikely, or if it be inoculated into guinea-pigs and transmit tuberculosis to the pigs, then the true nature of the lesion is certain.

(*q*) *Tuberculosis of the Spleen*

Tuberculosis of the spleen exists as part of miliary or generalized tuberculosis.

(*r*) *Tuberculosis of the Liver*

Tuberculosis of the liver is also possible, and can be suspected only when there is general tuberculosis.

(*s*) *Tubercular Pericarditis*

PERICARDITIS DUE TO TUBERCULOSIS occurs frequently. Only careful study of the case, the presence of tuberculosis in other portions of the body will help to make the diagnosis. The physical signs of simple and tubercular pericarditis are about the same.

B. Non-Bacterial Fungus Infection

THE MYCOSES

1. Actinomycosis

Organism.—Actinomycosis is a chronic infection due to Actinomyces bovis or ray fungus. It may affect practically any portion of the body.

Site.—This disease is common in cattle, and affects particularly the jaw. The fungus occurs in grain, and may be transmitted to man by the handling of infected grain. Infection takes place through the mouth or through an abrasion of the mucous membranes, or through an abrasion of the skin.

Incipient Stage.—In the beginning there is an indurated mass over the organ affected. This mass softens, ulcerates and discharges a semipurulent material through several sinuses. Examination of this material will show at once the large ray fungus, and the diagnosis can be made (Fig. 14).

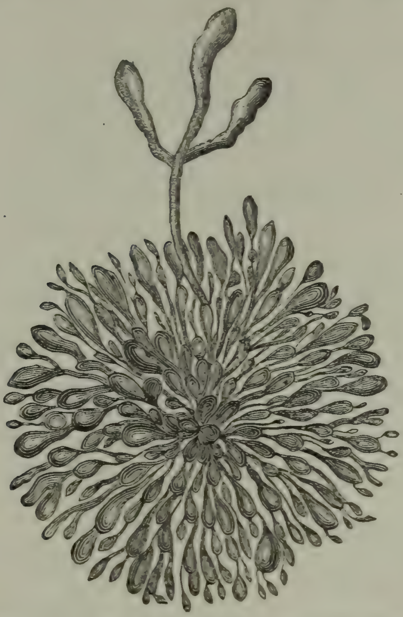

Fig. 14.—Actinomyces (Ray Fungus) with One Branching Filament Separated from the Others (Ponfick).

Diagnosis.—This disease affects internal organs such as the lungs, liver, and intestines. When the disease affects the internal organs, diagnosis of course is more difficult, and can only be made by *examination of some of the material causing the inflammation of the organ affected.* When the active organism is implanted about the face or neck, it most frequently takes its origin in the pharyngeal or buccal surface. There is a chronic suppurating induration, which is frequently the seat of discharging sinuses. The condition may be mistaken here for foci of tuberculosis, with unhealed sinuses, or for an old necrotic area about a carious tooth or in the shaft of a bone. Only the history of the case with its marked chronicity, and the demonstration of the infecting organism will make the diagnosis. It may also be mistaken for any new growth.

Diseases to be Differentiated from Actinomycosis

Pyemia
Tuberculosis
Empyema
Osteomyelitis
Cancer
Syphilis
Typhoid fever.

PYEMIA—TUBERCULOSIS.

When the condition is generalized with foci in various portions of the body, there is the picture of pyemia, with irregular temperature curves, and widely separated suppurating foci. The chronicity of the case separates it from the usual attack of pyemia, but makes it resemble a chronic case of tuberculosis. The presence of the ray fungus and the absence of tubercle bacilli separate it from either of these, and the presence of tubercle bacilli makes a positive diagnosis for tuberculosis.

EMPYEMA.

When the implantation is in the lung, there is a marked resemblance to tuberculosis of that organ, with fever; sweats; expectoration; and consolidation of the lung. The presence of the organism is the only positive sign. Certain cases resemble rather closely empyema. The writer has seen a case with a large amount of pus in the pleural cavity. A rib was resected, and the typical granuloma was found in the cavity of the chest. Here, too, the finding of Actinomycosis bovis is the only positive sign.

OSTEOMYELITIS.

When the bones are affected, the condition resembles an osteomyelitis, possibly tuberculous, or syphilitic in character. Here the Wassermann reaction will make the case syphilis, and the presence of actinomycosis will make a positive diagnosis of that condition.

CANCER—SYPHILIS—TYPHOID FEVER.

Intestinal actinomycosis results in a most varied set of symptoms, which may be mistaken for almost any inflammatory condition of the abdominal cavity, among which may be tuberculosis, cancer, syphilis and even typhoid fever.

The only possibility of a positive diagnosis is *recovery of the germ from some of the necrotic material,* the symptoms are so exactly the same. Of course the Widal reaction would be absent in the cases resembling typhoid fever, as would the Wassermann reaction in cases simulating syphilitic granulomata.

2. Sporotrichosis

Organism.—This is a chronic infection due to a parasitic fungus of the sporotrichosis group. The parasite affects human beings by accidental inoculation, through grains and fruit.

Various Forms of Parasite.—Osler describes in his book three forms. *The first* is a gummalike form occurring in subcutaneous tissue and in various portions of the body. They are small, round, solid nodules which break down; they form abscesses and ulcerate the skin.

In *the second form* it is ulcerative. On the hands and arms, the areas look not unlike cutaneous tuberculosis; however, they may occur on the lower extremities. They may be seen in groups and largely resemble syphilitic gummata.

In *the third form* there is a localized condition, a chancroidlike body, eroded on the surface. The skin draining, the part may become involved.

The fourth group are internal lesions of the mucous membrane of the muscles and of the joints.

Diseases to be Differentiated from Sporotrichosis

The diagnosis must be made from TUBERCULOSIS, SYPHILIS AND ACTINOMYCOSIS. *The diagnosis must always be made by culture, by agglutination and fixation reaction.* In no other possible way can one make a differential diagnosis from these various conditions.

3. Nocardiosis

Etiology.—Nocardiosis is a condition affecting the lungs, due to infection by a parasite which resembles both the bacteria and fungi. It resembles very closely pulmonary tuberculosis.

Diagnosis.—The diagnosis here also, can only be made by *examination of the exudate,* and *finding the organism present.*

Symptoms.—The symptoms are really those of a destructive disease of the lungs and pleura.

Diseases to be Differentiated from Nocardiosis

TUBERCULOSIS-ACTINOMYCOSIS.

The sputum of tuberculosis shows tubercle bacilli, the sputum of Actinomycosis, the spores and mycelium of that condition. The symptoms are those of actinomycosis.

4. Oidiomycosis

Organism.—Oidiomycosis is a form of infective dermatitis.

Site.—The skin lesion occurs on the face and resembles tuberculosis closely. Sometimes the granulomata break down and form little ulcerated surfaces. In a few cases the lungs and other parts have been affected.

Duration.—It is a chronic disease and may last for many years.

Diagnosis.—Here again the diagnosis depends entirely upon microscopic examination of the material from the small abscesses, or upon section of bits of excised skin.

5. Mycetoma

(Madura Foot)

Origin.—This disease is a chronic one due to implantation of a streptothrix, or growth resembling actinomycosis.

Symptoms.—It is characterized by swollen nodular groups and multiple abscesses. The nodules break down and form small abscesses, discharging remarkable black or yellow granules, one millimeter in diameter. These granules are composed of a vegetable parasite and its results (Fig. 15).

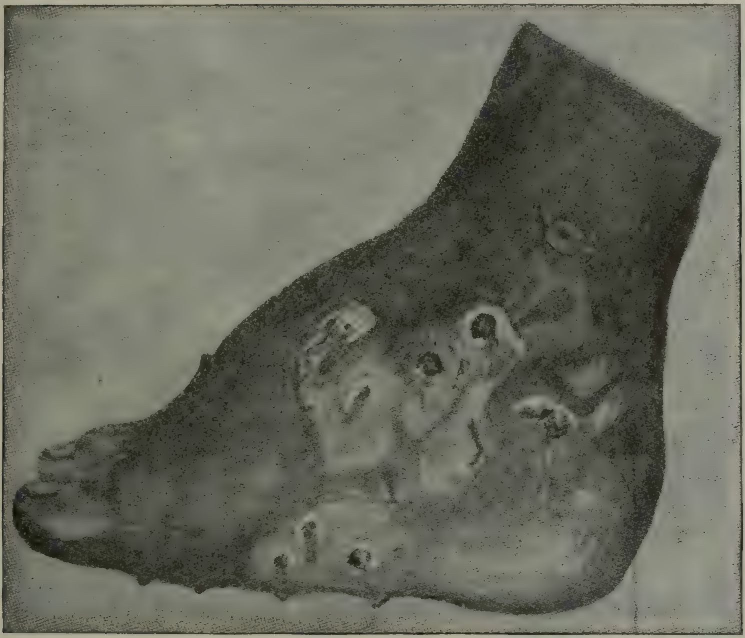

Fig. 15.—Mycetoma (L. Raynaud).

Course of the Disease.—The disease begins as a granuloma with a swelling of the foot, generally on the sole. The tumors soften; the foot increases in bulk and often becomes enormous in weight and size; numerous sinuses form, passing between the bones and discharging mucopurulent material. According to Wright and others the parasites are found in this discharge.

Place of Causative Organism.—There is still a difference of opinion as to the proper place of the causative organism among the fungi.

Conditions to be Differentiated from Mycetoma

The condition may be mistaken for Sarcoma or Syphilis.

SARCOMA.

Sarcoma is more acute; there are no real sinuses in the foot. Excision will show the characteristic round or spindle cells of sarcoma under the microscope. There are no spores or mycelium.

Syphilis.

Syphilis can be recognized by the history of an initial sore, by the presence of a Wassermann reaction and the absence of fungus spores or mycelium in lesion.

6. Aspergillosis

Organism.—This disease is due to the Aspergillus fumigatus (Fig. 16). It is a widely distributed parasite which is usually harmless. It gets foothold in the external auditory canal, genitals, eyes, etc.

Site.—The germ may cause, however, a disease of the lungs which closely resembles tuberculosis.

Symptoms. — There is cough, fever, expectoration; there may be cough without symptoms, as in the case reported by Osler.

Diagnosis. — The diagnosis can be made by examination of the expectoration, in which the germ can be found.

In examining the sputum it is well to mix it with 20 per cent solution of sodium hydrate, centrifugate, wash, and examine.

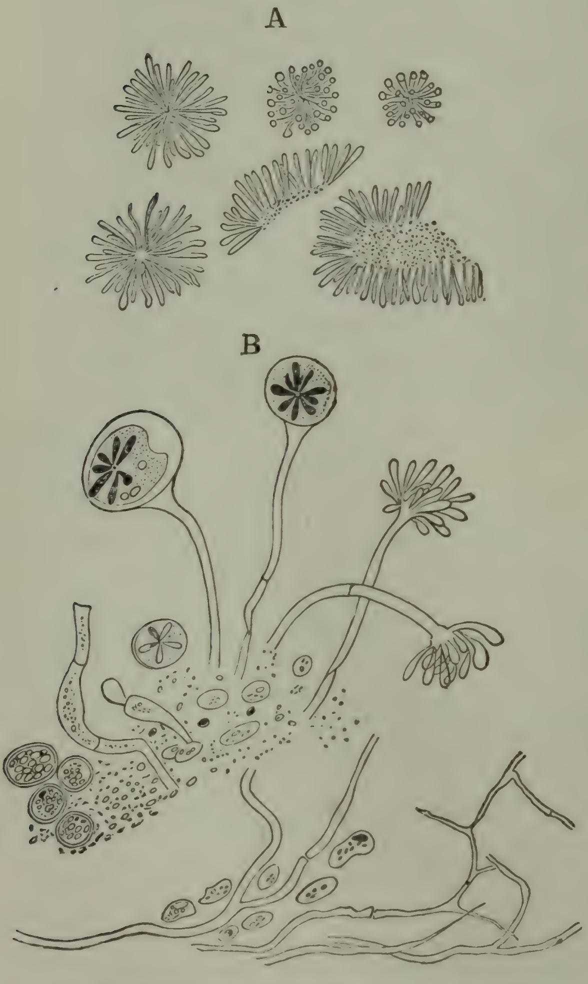

Fig. 16.—Aspergillus fumigatus (Fowler and Godlee, published by Longmans, Green & Co.).

Diseases to be Differentiated from Aspergillosis

Tuberculosis, bronchitis, emphysema.

The course of the disease and its physical signs almost exactly resemble Tuberculosis. *Examination of the sputum is the only possible way of differentiating.* In other cases there may be symptoms of a simple BRONCHITIS or EMPHYSEMA, both in reality existing, but the *presence of the fungus in the expectoration* will make the diagnosis plain.

C. Protozoan Infections

1. Psorospermiasis

Organism.—Psorosperms are widely spread throughout the invertebrate kingdom, but very uncommon in man. The only species which causes trouble in human beings is the *coccidia.* This coccodia has been found in the liver, the peritoneum, the omentum and in the pericardium.

Differentiation.—In addition to local symptoms, the case has the appearance of one of TUBERCULOSIS, the nodules being found in any part of the body. Examination, however, will reveal the coccidia and substantiate the diagnosis.

2. Amebiosis

(*Amebic Dysentery*)

Organism.—The Entameba histolytica and Entameba tetragena are the specific amebae of this condition.

Characteristic Features.—This is one of the tropical forms of dysentery characterized by bloody stools, tenesmus, fever, ulceration of the bowels, solitary liver abscess, and the presence of amebae in the feces.

The cases may be mild, moderate or severe, and may be gradual or rapid in their onset.

Mild Cases.—In mild cases there is weakness, lassitude, constipation and diarrhea alternating each other. These symptoms may last for many weeks before the patient realizes he is ill. Examination of the stool, however, will at once show the presence of entamebae (Fig. 17).

Neglected Cases.—If the case is neglected liver abscess may develop. A case of amebic liver abscess in a returned missionary was recently seen in which the patient, a physician, believed he had no dysentery.

Cases with Acute Symptoms.—Some cases begin suddenly, though Strong believes that some of the cases with acute symptoms have been in existence with few symptoms for a long period.

There is a sudden diarrhea with fifteen to twenty stools in the 24 hours, much abdominal pain, and leukocytosis; often death ensues.

Chronic Cases.—In chronic cases there are attacks when the patient is quite ill; there are other times when he is better. Some cases have a chronic dysentery.

Prominent Symptoms.—Diarrhea is intermittent. Blood, pus cells, shreds of tissue and amebae are present.

Complication.—Abscess of the liver is a common and serious complication. The abscess is usually unilocular.

Differentiation.—The disease may be mistaken for Other Forms of Dysentery. Some cases are complicated by bacillary infection, other cases have an innocuous form of amebae. The symptoms are the same; the diagnosis must be made by the microscope.

Methods of Diagnosis.—The following description taken from Strong, Osler & McCrae, Modern Medicine, can be depended on: "The examination of the stools should be made as soon as possible after they are passed, and the specimens should be collected free from urine. Many observers recommend in cold climates that a warm bed-pan be used and that the microscope slide be gently warmed. This is not necessary in tropical countries. These precautions are necessary because the amebae frequently die in the stools that have stood for any length of time or that contain urine, and their motility is often quickly impaired by cold; motile and resting amebae also quickly disintegrate in cold stools. The amebae must be found living and motile. In this condition they are easily recognized and cannot be mistaken for other cells. After movement ceases

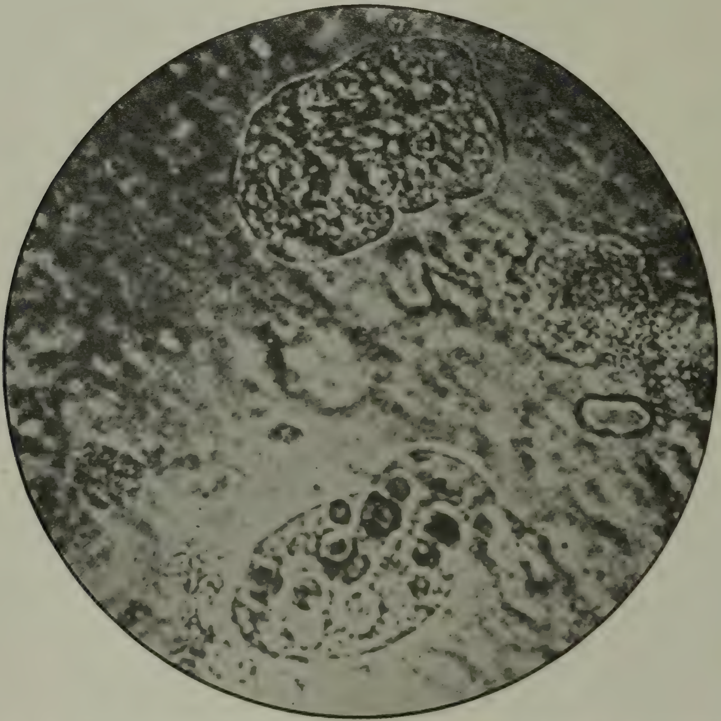

Fig. 17.—Entameba tetragena. (From Bull. No. 1, 1913, Office of Surgeon General.)

and death results, it is frequently impossible to distinguish them from other substances. If bloody mucus or small pieces of necrotic tissue are present, these should be examined first, for if they come from the neighborhood of an ulcer they usually contain very large numbers of amebae. If the movements are not liquid a dose of Rochelle salts should be given and the fluid portion of the stool examined. Another convenient method of securing material for examination is by the passage of the rectal tube. When the stools are fluid, considerable amounts may be obtained, or small portions of mucus will be found in the lumen of the tube. The occurrence of amebae in the tropics which responds to the hypodermic injection of emetin will separate the condition from any other form of dysentery. The abscess which follows or is a part of the disease is solitary. It may be confused with the abscess from infection of the abdominal cavity but in the latter there are signs of existing or previous appendicitis or bile duct disease, and the abscesses are multiple."

3. Malarial Fever

This condition—malarial fever—as is now well known, is due to infection by Plasmodium malariæ, an hematozoön which infects man. This infective organism is carried from an individual suffering from malaria to a healthy individual by a species of mosquito, the anopheles. These organisms can be demonstrated either in the fresh blood or in a dried specimen stained by any one of the proper blood stains.

It is one of the *infectious diseases.* Again the fact should be accentuated that every practitioner of medicine should be familiar with laboratory methods. He should not only be familiar with them, but he should put them into practice, either by his own use of them or by employing some one to do this routine work.

Types of Malaria.—Three distinct types of malaria are known, due to infection by different species of this organism: the *tertian,* the *quartan* and the *estivo-autumnal.*

MALIGNANT MALARIAL FEVER is the result of infection by the *estivo-autumnal type* of organism which occurs usually in the tropics or subtropic regions, and differs in many of its symptoms from the tertian type seen in temperate regions.

VARIETIES.—The *comatose* and *algid* forms are also two very fatal varieties.

GRADE OF PERIODICITY.—These types get their names by reason of the grade of periodicity in each. The *tertian* is characterized by a paroxysm, later to be described, occurring every other day. The paroxysm of the *quartan* variety occurs every fourth day. The *estivo-autumnal* paroxysm occurs at rather irregular periods, and sometimes the fever is continuous.

A double infection by the tertian organism—that in which the individual is infected by two different sets of organisms which run their course each in three days, maturing on alternate days—gives rise to the *quotidian form* of malarial fever.

Organism.—The organism which is the cause of this heretofore destructive disease gives opportunity for one of the most entertaining studies in medicine. The organism of the tertian variety is the smaller, the quartan larger, the estivo-autumnal different in certain particulars.

Tertian Parasite.—The tertian variety of the organism, Plasmodium vivax, in the earliest stages is a small ameboid hyaline body within the red corpuscle, which is with difficulty distinguished in the living blood. Soon, however, the cell infected with the organism begins to lose its pigment, by reason of the fact that the organism absorbs it. Then the body of the organism is rapidly filled with the pigment granules and becomes easily visible. The pigment takes on a brownian movement—this adds an additional method of identification.

Meanwhile the infected cells become gradually paler, swell and are distinctly larger than the rest of the corpuscles in the field; the pigment becomes concentrated in the center of the ameboid body, and the protoplasm of the body arranges itself around this central pigmented mass and finally divides into fifteen or twenty ovoid bodies. The shell of the red corpuscle disappears. There then remains a rosette body with the detached pigment as its center with the oval bodies formed from the protoplasm of the plasmodium arranged around it. The ovoid bodies then separate, to again enter the red corpuscle.

Quartan Parasite.—The quartan parasite is larger, the pigment coarser and the ovoid bodies of the rosette fewer in number.

Estivo-autumnal Parasite.—The estivo-autumnal plasmodium is smaller, the pigment scarce and the rosettes exactly circular. After a time crescentlike organisms with central pigment form, which are characteristic of this type. The gametocytes or sexual forms are developed later; the flagellae are a part of this stage of the organism.

Mode of Transmission.—From the body of an infected human being the mosquitoes of the species anopheles suck the infected blood; then the elements which evolve the flagellae go into complete sexual evolution in the body of the mosquito. The male and female unite and form a young plasmodium again which is found largely in the salivary glands of the mosquito. From this salivary gland the young plasmodium is injected into the blood stream of the human being by the bite of the insect.

Symptoms of Ordinary Simple Paroxysms of Malarial Fever.—The Paroxysm.—Preceding the actual paroxysm of the fever are often feelings of malaise and weakness. The symptoms of the attack occur usually at the time of the sporulation of the plasmodium.

Cold Stage.—The first sign is a severe chill. The features become

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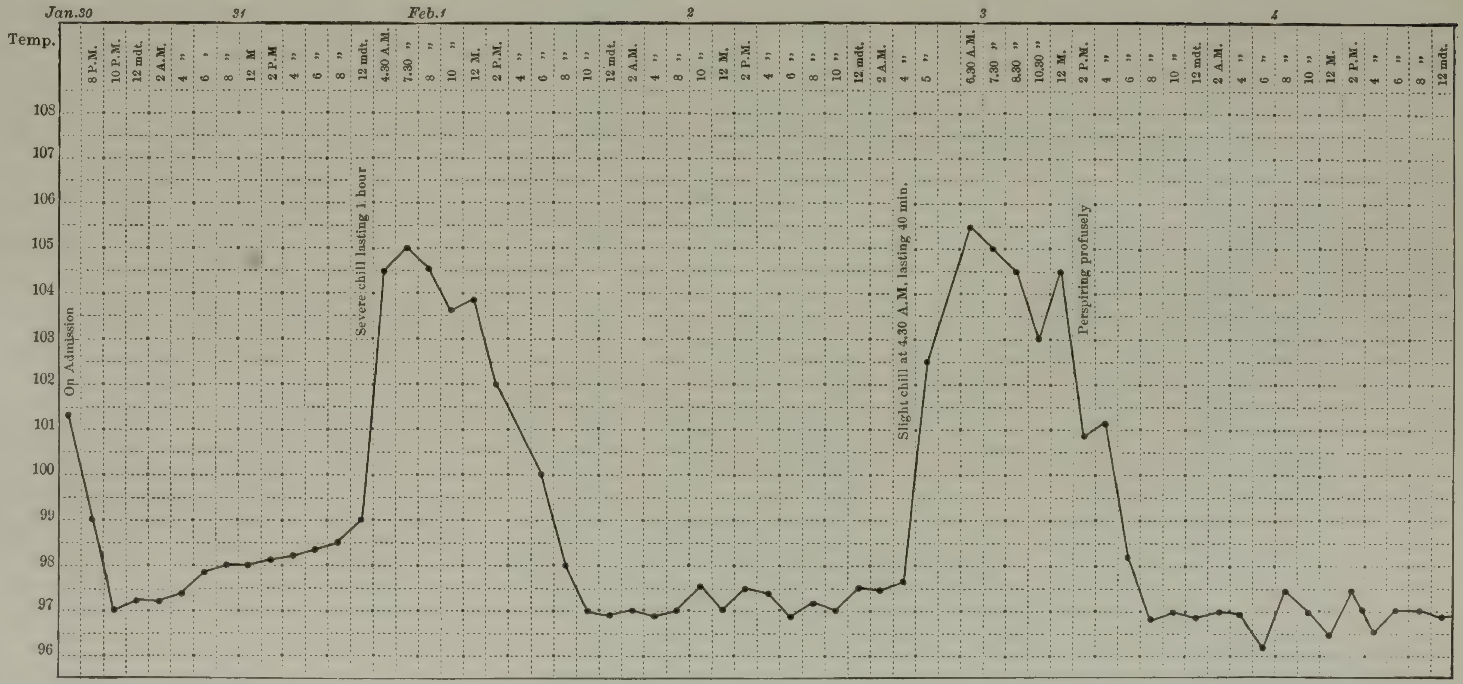

Fig. 18.—Chart of Tertian Malarial Fever. (From Osler's Practice of Medicine, 1898 ed.)

pinched; the hands are cold; the teeth chatter; the individual feels as though he would die of cold; often there is vomiting and there is severe splitting headache.

Fever Stage.—Even though the patient feels cold and shivers, accurate temperature records taken by the rectum will show a fever even at the beginning of the chill. After the cold stage has lasted from three to five minutes to half an hour, the temperature rises rapidly to 103° or 104° F., the patient feels hot, the headache becomes worse, the skin dry, and the mouth parched.

Sweating Stage.—After from one to two hours of this stage of fever the patient begins to sweat, beads of moisture stand out over the face and the sweat becomes so profuse over the whole body that the clothing is soaked.

Duration of Paroxysm.—The entire paroxysm lasts from two to four hours, leaving the patient ill, weak and exhausted. After the first paroxysm, however, the patient rapidly recovers, and in a few hours feels well (Fig. 18).

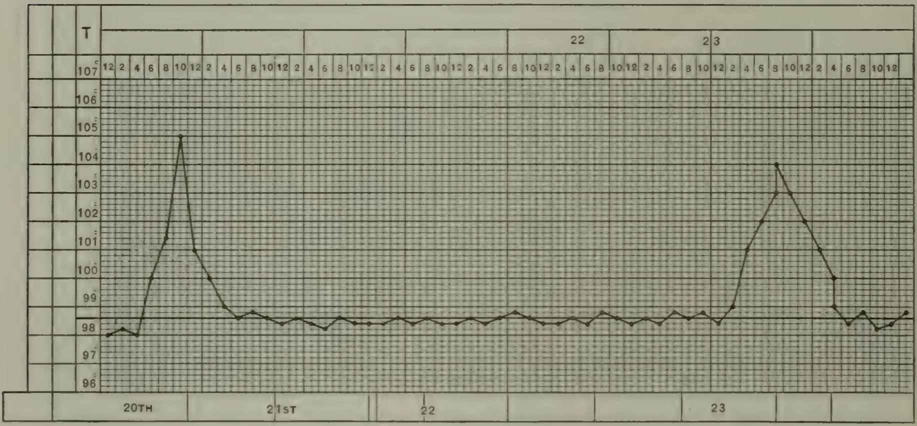

Fig. 19.—Chart of Quartan Malarial Fever. (Osler's Practice of Medicine, Edition 1912.)

Repeated Paroxysms.—In about forty-eight hours following, beginning at a time one to two hours earlier than the first paroxysm, the same series of events recurs. Repeated paroxysms leave the patient ill, anemic, weak and listless.

If the infection be of the *quartan variety*, the paroxysm will not recur until seventy-two hours after the initial one (Fig. 19).

In the *estivo-autumnal infection,* the chills are irregular as to time; sometimes the infection is so frequent and so intense that the fever is actually continued or remittent (Fig. 20).

When there is a *double infection with the organism of the tertian type* of fever, a chill occurs every day (quotidian type. (Fig. 21.)).

If there be a *triple infection by the quartan type* of Plasmodium malariæ, there will be a chill every day.

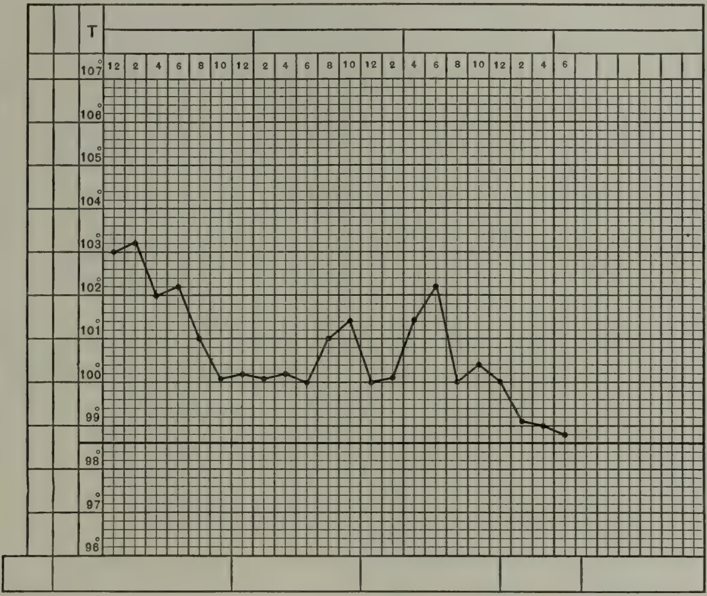

Fig. 20.—Chart Showing Curve of Fever in Autumnal Malarial Fever. (Osler, Edition 1912.)

Symptoms of Malignant Types of Malarial Fever.—Geographical Distribution.—Malignant malarial fever occurs in the tropics or in the southern parts of the temperate zone.

The Paroxysm.—In these severe forms of infection there is a sudden attack of extremely high fever, the patient becomes unconscious almost immediately, and there may be hemiplegia.

Site.—Here the organism may not be found in the peripheral blood, but it may be found in the blood of the splenic pulp. In these extreme types careful *microscopic diagnosis* is of the utmost importance.

The **algid form** is also malignant in type.

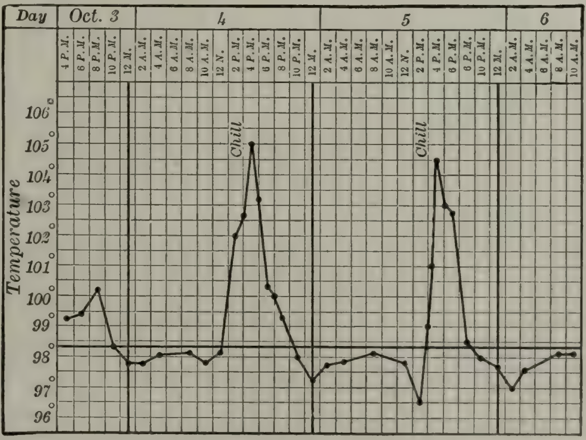

Fig. 21.—Double Tertian Infection—Quotidian Fever. (From Osler's Practice of Medicine.)

Here the patient is collapsed; there is a continued and lasting cold stage, the patient frequently dying before a diagnosis is made.

There is never leukocytosis in an uncomplicated case of malarial fever. When malarial fever becomes chronic there is a sallow appearance of the individual, and he becomes anemic and the spleen enlarges.

Diagnosis.—The diagnosis of malarial fever depends upon two procedures. A fair opinion may be formed when the symptoms are well defined and sharply marked, but the ultimate diagnosis is by the *microscope and by the use of therapeutic test.* Careful examination of a smear of the peripheral blood at about the time of the paroxysm will show the plasmodium mobile in the blood, or a specimen stained with Wright's stain will show a blue irregular body within the red corpuscle with pigment frequently in the center.

A true malarial fever will sooner or later—and usually in a characteristic manner—respond to the administration of quinin. The paroxysms will disappear in mild cases after the blood is saturated with quinin. In order to get this effect, the patient must be cinchonized between the end of one paroxysm and the beginning of the next.

Conditions to be Differentiated from Malarial Fever

The conditions with which malarial fever is most commonly confounded are:

Typhoid fever
Suppuration in some spot not easily discovered
Tuberculosis
Yellow fever
Unconscious states
Gall-stones
Weil's disease
Leukemia
Splenic anemia
Uncinariasis.

Typhoid Fever.

Typhoid fever usually begins with malaise and uncomfortable feelings, the temperature being about one degree higher each day until the fastigium is reached at the end of the first week. The temperature then remains about the same for two weeks and gradually falls to normal. In malaria, as has been described, there is a sudden rise of temperature and a likewise rapid fall, repeated at stated intervals. Typhoid fever is a long-continued fever with rose spots, enlarged spleen, diarrhea, abdominal distention, a peculiar coated tongue, and more or less delirium.

PLATE III

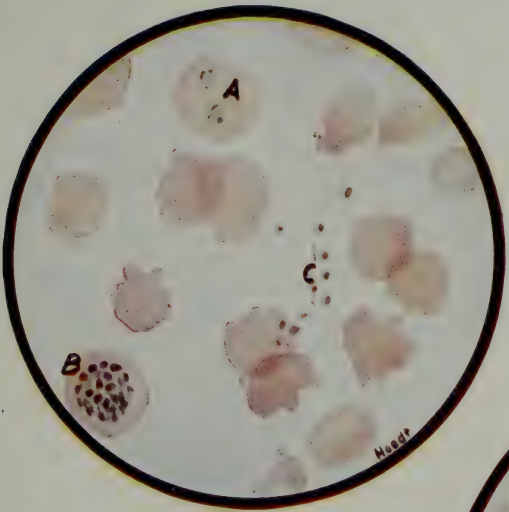

Fig. 1.

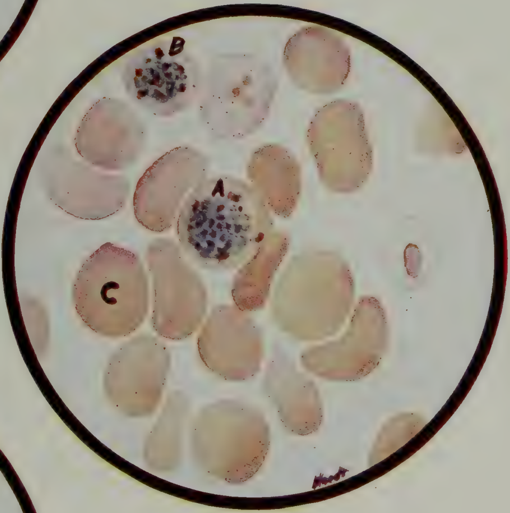

Fig. 2.

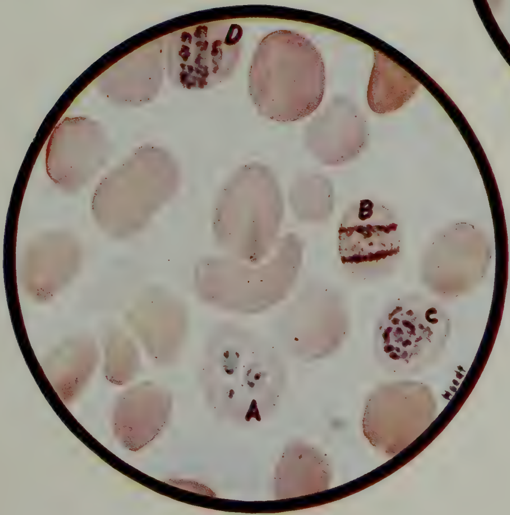

Fig. 3.

Fig. 1.—Plasmodium falciparum (Estivo-autumnal Parasite); A, Young Parasites—So-called Ring Form; B, Sporulating Parasite; C, Free Spores or Merozites.

Fig 2.—Plasmodium vivax (Tertian Parasite); A, Sporulating Parasite; B, Sporulating Parasite.

Fig. 3.—Plasmodium malariae (Quartan Parasite); A, Small Parasites—So-called Ring Form; B, Half-grown Parasite—Band Form; C, Three-quarters-grown Parasite; D, Large Band Form.

(Adapted and Colored from Bull. 1, Jan., 1913; War Department, Surgeon-General's Office.)

In estivo-autumnal fever the temperature is irregularly intermittent and remittent, but is never continuous as in typhoid.

In typhoid there is Widal reaction. It is absent in malarial fever. Malarial fever shows plasmodium in the blood, while typhoid fever does not.

Typhoid and malarial fever may affect the same person at the same time, when one disease masks the other, but there is no reason to long continue in doubt, if the *blood is examined.*

Toward the end of certain cases of typhoid fever, the temperature takes on a true intermittent type, which may be mistaken for malarial fever; there is little excuse for this mistake, however, in view of the previous history of typhoid and the absence of malarial organisms from the blood.

SUPPURATION.

Suppuration may frequently be characterized by a chill, fever and sweat very like a malarial paroxysm; this chill, fever and sweat may recur. However, these paroxysms are much more irregular as to time of occurrence than in malarial fever, and they may occur more than once a day. *There is always a leukocytosis in suppuration; there is never leukocytosis in malaria.*

Frequently in suppuration there is *some point of pain or tenderness* over some portion of the body, which is wanting in malaria. Usually a *blood culture* will show the infecting organism in suppurative diseases.

This septic infection has two particular localizations where the infection may be taken for malaria: LIVER ABSCESS and ENDOCARDITIS.

IN LIVER ABSCESS there is a history of amebic dysentery; the liver is enlarged and tender and the patient may be jaundiced. There is *leukocytosis,* however, in liver abscess—it is not present in malaria. The amebae can almost without exception be found in stools of amebic dysentery, which so frequently accompanies liver abscess. There is *no plasmodium* in the blood.

ENDOCARDITIS has many resemblances in the temperature chart to that of malaria, but the exacerbations of fever are irregular. There is *leukocytosis* and examination of the heart will show murmurs or *other signs of cardiac involvement.* There is an *absence of plasmodium* in the blood, and a blood culture frequently shows the infecting organism.

TUBERCULOSIS.

Tuberculosis is frequently characterized by an *evening rise of temperature* falling to or near normal in the morning, and it is an unfortunate fact that this fever is still often diagnosed as malarial fever. Failure of careful examination is the cause of the mistake. A failure to differentiate this condition is inexcusable.

In tuberculosis there is always a lesion in some portion of the body which can be distinguished. Usually this seat is in the lungs, but it may be in other organs. The fever of tuberculosis, while it is often intermittent, is not preceded regularly by a chill, and the rise of temperature usually appears in the evening. There is usually a cough. Examination of the sputum will show tubercle bacilli, and examination of the blood an absence of malarial organisms.

YELLOW FEVER.

Yellow fever has many of the symptoms of certain cases of malarial fever, but the *jaundice* and the *nephritis* of yellow fever mark the case as one of that disease. Certain cases of malarial fever of remittent type (estivo-autumnal infection) are marked with both jaundice and nephritis. Here the careful blood examination is the only positive sign, and should be availed of to make the positive differential diagnosis.

APOPLEXY—UREMIA—DIABETIC COMA—SUNSTROKE.

Any of the states which are accompanied by unconsciousness can be mistaken in malarial districts for the comatose forms of malaria. Apoplexy, uremia, diabetic coma and sunstroke are among these conditions. Careful and repeated blood and urine examinations will make the diagnosis.

In *apoplexy* there is paralysis of one side of the body.

In *uremia* albumin and tube casts can be found in the urine.

In *diabetic coma* there is air hunger; the ketone bodies together with sugar can be found in the urine.

Sunstroke is characterized by high fever.

In none of these can malarial organisms be found in the peripheral blood or in the blood of the spleen.

GALL-STONES.

Gall-stones in the common duct, especially those of the ball valve variety, have chill, fever and sweats as part of their symptomatology (the so-called Charcot's intermittent fever). Following or accompanying the attack there is pain and tenderness in the epigastrium; there is likely to be much jaundice following the attacks. The latter are irregular in their occurrence. There are no malarial organisms in the blood.

WEIL S DISEASE.

Weil's disease, or infective jaundice, may possibly be mistaken for the condition, but the blood examination will make the diagnosis positive.

LEUKEMIA, SPLENIC ANEMIA.

By reason of the increasing anemia and enlarged spleen in chronic malaria, the condition might be mistaken for leukemia or splenic anemia.

In LEUKEMIA there is leukocytosis and absence of malarial organisms.

In SPLENIC ANEMIA there is progressive anemia, pigmentation of the skin and at times irregular fever. The differentiation must be made by the absence of the malarial organism and by the history of the case. There is an entire *lack of the regularly intermittent fever in splenic anemia.*

UNCINARIASIS.

The weakness, sallow color, and emaciation common in this disease are mistaken without any reason for malaria. Examination of the blood will not show the malarial organism, and examination of the stool will show the ova or the adult of the hookworm.

Typhomalarial Fever a Misnomer

The name typhomalarial fever is a misnomer. Much harm has been done and many lives lost by its false conception. Typhoid fever and malarial fever may exist in the same individual at the same time, and should be diagnosed, but the condition is a mere accidental infection; there is no such entity as typhomalarial fever.

4. Trypanosomiasis

(*Sleeping Sickness*)

Geographical Occurrence.—Trypanosomiasis is a chronic, infectious disease occurring in Africa, affecting the natives by preference.

Organism.—It is caused by a flagellate infusorium—Trypanosoma gambiense and rhodesiensi. It is introduced into the blood of human beings by the bite of the tsetse fly, the Glossina palpalis and Glossina morsitans. Authors describe the fly as living in certain zones upon the bushes and feeding upon the blood of antelopes and crocodiles. The trypanosome undergoes changes in the body of the fly; the insect does not become infective for thirty-two days.

Symptoms.—*First Stage.*—The symptoms of the disease in the first stage are occasional fever with a very rapid pulse, also puffiness of the face. There is always a general adenitis, the enlarged glands containing trypanosomes. Bruce believes that while fever occurs, it is by no means universal. During the time of the general adenitis the natives go about without symptoms; in Europeans the quiescent stage is not so marked. There is irregular fever, enlarged spleen, and sometimes erythematous spots over the body.

Second Stage.—In the second stage, that of SLEEPING SICKNESS, the patient first becomes dull and apathetic. He is dull-eyed, has headache, indefinite pains over the body, with rapid pulse; the gait is weak and shuffling; there are tremors and weakness; fever is irregular. The voice is weak, the patient sleeps well. There is always enlargement of the lymphatic glands; the heart is always weak.

Diagnosis.—Diagnosis of this condition to the uninitiated is difficult, but given a patient who has been exposed to the bite of the tsetse fly, with fever, dullness of the intellect and emaciation, the disease would be suspected. This of course can be confirmed at once by examination of the blood, in which the trypanosome will be found. The organism can always be found in the blood or spinal fluid, or as described by Bruce, puncture of a lymphatic gland is quite as diagnostic.

Conditions to be Differentiated from Trypanosomiasis

The disease may be confounded with:

Beriberi
Nephritis
Intracranial Syphilis
Brain Tumors
Tabes
Malarial Fever.

BERIBERI.

Beriberi, which is a tropical disease, but not African, can be traced to the eating of polished rice. It has as its characteristic symptom persistent neuritis. It is also an afebrile condition. There are no trypanosomes in the blood.

NEPHRITIS.

From nephritis it can be distinguished by examination of the blood and glandular juice which will not show the organism, and by the typical signs of nephritis.

INTRACRANIAL SYPHILIS—BRAIN TUMORS.

The condition might be mistaken for intercranial syphilis or tumors of the brain, but here the typical conditions of a nervous lesion, the absence of the blood findings, and the absence of a temperature, will make the diagnosis.

TABES.

In tabes and general paralysis of the insane, there might be some difficulty in making the diagnosis certain, either positively or negatively, but again the blood picture is entirely different from that of trypanosomiasis and the Wassermann reaction can be found in the blood or spinal fluid.

MALARIAL FEVER.

The irregular fever may be suggestive of malarial fever, but in malaria there are plasmodiae and not trypanosomes, and the administration of quinin will control the fever.

5. Kala-azar

(*Leishmaniasis*)

Organism.—This disease is due to a protozoan infection—the Leishmania donovani which lives in the cells of the spleen and bone marrow. It is common in many regions of the Orient.

Mode of Transmission.—Its mode of transmission is not definitely known, but it is probably spread by some plant feeding bug or the bedbug.

Period of Incubation.—The period of incubation is not certain.

Symptoms.—This is a disease characterized by intermittent or remittent fever, marked emaciation, and enlargement of the liver and spleen.

Course of the Disease.—It begins with chills; there is fever rising to 104-106° F., vomiting, enlargement of the liver and spleen. The fever is irregular in type, remains high for a few days or six weeks, to be followed by a low grade continuous fever.

There is debility, emaciation and anemia; the skin is pigmented and of a gray drab color; there are subcutaneous hemorrhages. This may last for several months, or it may be prolonged for two years. Enlargement of the spleen is a continuous factor.

Leishmania infantum.—There is a form of leishmaniasis common along the Mediterranean which affects *infants* almost exclusively. The infection is through the Leishmania infantum. This form of the disease resembles the ordinary splenic anemia of children. The diagnosis must be made from splenic anemia.

In splenic anemia there is pigmentation of the skin; the spleen is enormous in size and there is a tendency to hemorrhage. There is a tendency in later stages to cirrhosis of the liver. No organism can be demonstrated in the blood.

6. Relapsing Fever

(*Febris recurrens*)

Organism.—This is a specific fever caused by different varieties of spirochetes.

Clinical Picture.—The clinical picture is essentially the same in all the varieties, though in African tick fever the prognosis is grave.

The African relapsing fever is also due to a spirochete, and is spread through the agency of a tick Ornithodorus moubata.

Geographical Distribution.—This fever is not common in either England or America, but is quite common in India.

Mode of Transmission.—It is spread by means of ticks inhabiting the various regions infested with the disease. Todd believes that the usual method of transmission is through lice infesting an infected individual.

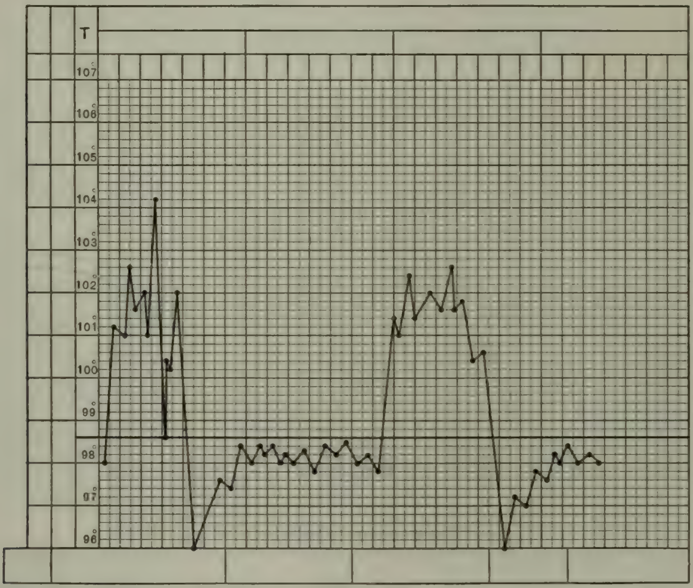

Fig. 22.—Chart of Relapsing Fever. (From Osler's Practice of Medicine.) (After Murchison.)

Symptoms.—The symptoms of the disease are quite characteristic. After a few days of invasion, there is an abrupt chill immediately followed by fever, nausea, pain in the back muscles, headache, vomiting, and sometimes by convulsions.

The fever ranges from 102° to 105° F., lasts from four to six days, then falls abruptly to normal, accompanied by marked sweating. The patient often seemingly recovers and can move about, but in the course of a week or ten days there is a relapse (Fig. 22), when the spirillum is again found in the peripheral blood.

During the fever there is enlargement of the spleen; jaundice and digestive symptoms often occur. In very rare cases—eight out of six hundred (Murchison)—there is a slight rash resembling measles. Leukocytosis is the rule, most marked after the crisis (Cabot).

Diagnosis.—An absolute diagnosis can be made by finding spirochetes in the blood. A modified agglutinin reaction (after Lowenthal) may be made in the case of a suspected individual, by mixing a drop of blood in which spirochetes cannot be demonstrated, with a drop of blood containing spirochetes. If the case is one of relapsing fever the spirochetes will be clumped after being half an hour in the incubator.

Conditions to be Differentiated from Relapsing Fever

The disease may be mistaken for:

Typhoid fever
Typhus fever
Pneumonia
Smallpox
Cerebrospinal fever
Malarial fever
Influenza.

The differentiation must depend upon laboratory methods, physical signs, and lapse of time; usually laboratory methods and physical signs will suffice. In other cases of suspected relapsing fever the blood should be examined for the spirochetes early; a blood count should be made, and an agglutinin reaction done during the apyrexia. If the spirochetae are present, and if the serodiagnosis is positive during the apyrexia, the diagnosis may be made at once as relapsing fever.

TYPHOID FEVER.

This disease begins gradually; a fall of temperature lasting several days is not symptomatic of typhoid fever. Diarrhea, common in typhoid fever, is not characteristic of relapsing fever. The serum reaction with typhoid bacilli is present in typhoid fever; it is absent in relapsing fever; there is a leukopenia in the former. As a rule care must be taken, for in some rare cases of typhoid there is a leukocytosis in the very beginning. Spirochetes cannot be demonstrated. A blood culture will show the presence of Eberth's bacilli in typhoid fever.

TYPHUS FEVER.

Here the beginning more closely resembles relapsing fever, but there is no abrupt crisis to be followed by fever in eight to ten days. The petechial rash of typhus is more or less characteristic. There is seldom a rash in relapsing fever; when it occurs it resembles measles rather than a true petechial rash.

Pneumonia.

Here there is the same rapid rise of temperature, but there is a characteristic chill. Leukocytosis appears in the very beginning. Careful physical examination will usually reveal signs in the chest, limited motion, dullness, difference in breathing in the two sides, etc.

Herpes is also quite common in pneumonia. The sudden full temperature is usually followed by a fall in the number of leukocytes and not an increase, as in relapsing fever. Spirochetes cannot be demonstrated in the blood.

A blood culture will show pneumococci in certain cases of pneumonia, and pneumococci can be demonstrated in the sputa.

Smallpox.

After the third or fourth day there is the characteristic papular rash on the face, bust and hands. Even in the hemorrhagic forms the rash is papular and not macular as in relapsing fever. Of course the spirochetes are absent.

Cerebrospinal Meningitis.

Cerebrospinal meningitis might be mistaken for relapsing fever, but the muscle contractures, the hyperesthesia, the ocular symptoms—all go to suggest meningitis. A spinal puncture will, in meningitis, show a cloudy fluid in the septic forms, such as epidemic cerebrospinal meningitis, pneumococcic, streptococcic; tubercle bacilli may be demonstrated in the clear fluid of tubercular meningitis.

All of the above conditions can be differentiated by waiting for the crisis of relapsing fever and for the relapse.

Malarial Fever.

Malarial fever might be mistaken for relapsing fever, but the shorter course of the pyrexia, or the greater irregularity of that course, together with plasmodium in the red cells will make the diagnosis.

7. Syphilis

Organism.—Syphilis is due to infection with the Spirochetæ pallida or Treponema pallidum (Fig. 23).

Forms of Syphilis

There are two forms of the disease, (a) *acquired* and (b) *hereditary*, each having many different symptoms.

(*a*) *Acquired Syphilis*

Origin.—Acquired syphilis is transmitted by actual contact of the sick with the well, usually by sexual intercourse—and in the great majority of cases by illicit sexual intercourse—though there are extragenital means of transmission, such as kissing.

First Stage.—The first symptom of acquired syphilis is the *chancre*, or primary sore. This may occur at any portion of the body, but from the nature of the transmission it is usually about the genitals. Extragenital chancres may appear on the tongue or lips, transmitted by improper practices or by kissing; it may occur on the fingers or hands of a surgeon by reason of infection received in the course of professional work. It is often difficult to distinguish, especially in females, when not ulcerated, or when situated in the urethra. Its one characteristic is hardness. Much induration of a venereal sore causes grave suspicion that it is syphilitic. It is rarely multiple but may be. Often it is not ulcerated, especially in the early stages, when it has the appearance of a papule from which the top layers of the epidermis have been rubbed. Spirochetes can be demonstrated in the exudate or sections of a chancre. With the appearance of the chancre, the lymph glands in the region begin to enlarge in the groins, if the chancre is situated about the genitals; in the submaxillary region if the sore is about the mouth or tongue. Soon the entire glandular system becomes involved; the posterior cervical and epitrochlear are the most easily felt and should be searched for.

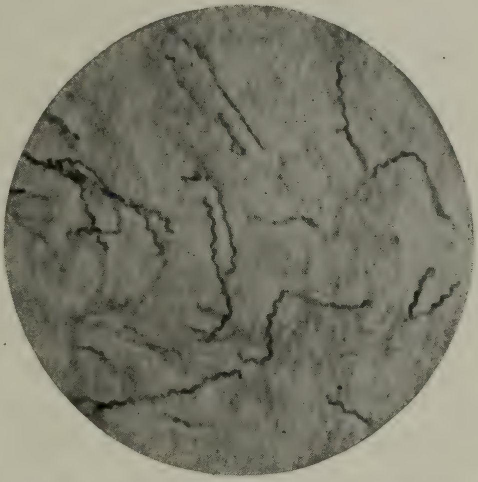

Fig. 23.—Treponema pallidum. (From Bull. No. 1, Med. Dept., U. S. A., 1913.)

Second Stage.—Soon after the beginning of the chancre, the patient becomes ill, with *general pain, fever, weakness and emaciation,* a condition which is difficult to diagnose if the history is not accurate, or if the chancre cannot be found.

In from four to six weeks, a *rash* appears. This rash may be a simple roseola, unnoticed by the patient because it does not disturb him; it is less common on the face than upon other portions of the body; or the rash may be covered with scales, the squamous syphilide; it may be papular and resemble smallpox in its first appearance; or it may be pustular, again being confused with smallpox or chicken-pox. The scars from this rash are frequently copper colored, which help much in the later diagnosis.

With the appearance of the rash the *hair begins to fall out*—great bunches may be detached from the head by merely grasping the hair with the fingers. The eyebrows fall, and the condition may advance to a complete alopecia.

The *nails become brittle* and may even become detached from the matrix. In the mouth, about the anus and in the groins, ulcerations or flat tumors with a moist surface covered with a grayish exudate may occur. These are the *mucous patches.* Iritis is common and may be recognized by its periciliary injection, pain in the eye made worse by light and by fixation of the iris.

Anemia is common and frequently extremely severe, or it may be a slight chloranemia.

Tertiary Stage.—The tertiary stage begins from six months onward after the appearance of the chancre. Deep ulcerations of the skin occur, which are frequently symmetrical. Osteitis in various portions of the body, particularly in the nasal and palatal bones, also occurs. This causes the terrible deformity too often seen in which the whole bridge of the nose and palate have disappeared and a great hole occurs where the nose should be. When the septum has disappeared without destruction of the soft parts, the so-called saddle-back nose appears. The peculiar "nasal" tone of the voice occurs in these conditions.

At this time various internal organs and the nervous system may be affected, causing the diseases peculiar to these organs. The liver is especially susceptible. Here there may be gummatous masses appearing as tumors of the liver, or perihepatitis, or scarring of the liver may occur, the latter causing a lobulated liver.

Diagnosis.—Diagnosis can now be made early. Expert examination of scrapings of a suspected sore will invariably show the spirochetae if the case is one of syphilis. A dark field microscope is essential for this work. An early and efficient treatment before the deep organs have become invaded by the organism will prevent serious complications and the disease will be much shortened. This is a most important measure.

The Wassermann reaction must be made use of. Eighty to ninety per cent of syphilitic cases give a Wassermann reaction. In cases of tabes dorsalis, tapping the spinal canal and testing the spinal fluid for the complement fixation is most necessary. Luetin, a preparation of treponema pallidum, causes a skin reaction comparable to that of tuberculin. Careful examination for chancre, for old scars, for contractions in the throat, colored scars, enlarged liver, and high blood pressure, will help make the diagnosis.

(*b*) ***Hereditary*** (***Congenital***) ***Syphilis***

Symptoms.—Hereditary syphilis is characterized by changes in the internal organs, the spleen, liver, etc., by inflammatory conditions of the serous membranes, and if the child live, by stunted growth and badly formed characteristic teeth, etc.

Origin.—A child may be born with a syphilitic rash and run through all the stages of acquired syphilis, or it may be born apparently healthy and after a few weeks develop signs of syphilis. These forms of active hereditary syphilis are just as contagious as the acquired forms. A child may acquire syphilis from its father or from its mother, one of the two being healthy, or both father and mother may be affected. A healthy mother may bear a syphilitic child, the child being infected from the father. Under these conditions the mother becomes immune to syphilis though she may show no sign of the disease and cannot be infected by her child who can infect any other healthy individual (Colles' law).

Diagnosis.—**Congenital syphilis** is characterized by symptoms which occur at birth and by others which occur later. The syphilitic infant is ill-developed and may have a rash. Bullae, etc., are over the body; there are snuffles; the angle of the lips is fissured.

If the condition develops later, snuffles, keratitis and rhagades occur. The fingers become enlarged and onychia occurs; the spleen is often enlarged; sometimes hemorrhage occurs. Still later, bone lesions and keratitis or iritis are present.

In all these cases Wassermann reaction will clear up a diagnosis. The enlarged spleen might be mistaken for that of leukemia. The blood, however, will not show any leukemic changes. Keratitis may be confounded with tubercular keratitis.

Cerebral Syphilis

Symptoms.—Syphilis of the brain usually gives the symptoms of a tumor, headache, dizziness, delirium, papillo-edema, possible local paralysis of certain groups of muscles or a sudden hemiplegia. Owing to the cerebral arteritis which frequently exists, there may be a rupture of a blood vessel, bringing on a complete hemiplegia. A hemiplegia before thirty-five or forty years of age is extremely suggestive of syphilis.

Tabes may be the end result. The sclerosis of the posterior columns of the cord comes on insidiously, with failing sight, gastric crises and failing sexual powers as early symptoms.

Rapidly failing sight must lead to a careful examination of the eye grounds, as one of the very first measures. Attacks of sudden, unaccountable vomiting and depression in a young individual must be a reason for a careful examination of the nervous system in all such persons. More than one individual has had the abdomen opened for pyloric stenosis, when gastric crises of tabetic origin were the real cause.

Method of Diagnosis.—Syphilis of the cerebral system may be diagnosticated after the methods of Swift and Ellis. The spinal canal is tapped; the cells are counted in a blood counting chamber after being diluted with *one* part of a 10 per cent solution of acetic acid. If the cells are above six to the cubic centimeter it shows an irritation of the cerebrospinal system. If the fluid gives the Wassermann reaction, the inflammation is due to syphilis. The fluid will also be rich in globulin, will reduce Fehling's solution and may also give the colloidal gold test, after the method of Lange.

Conditions to be Differentiated from Syphilis

Syphilis may be mistaken for:

Chancroids
Venereal warts
Typhoid fever
Malarial fever
Exanthemata
Rheumatic fever
Psoriasis
Malignant disease of the liver
Carcinoma of the liver
Cirrhosis of the liver
Multiple neuritis
Tuberculosis.

CHANCROIDS.

This is a venereal inflammation usually on the genitalia. The sore is painful and ulcerating; it appears immediately after infection and may be multiple. The bubo in the groin is likely to suppurate—this is unlike the chancre described above. Spirochetes cannot be demonstrated in the exudate from the sore. Chancroids are not followed by rash, sore throat or other secondary conditions; only the inguinal gland is infected; there is not multiple lymph infection.

VENEREAL WARTS.

These are hypertrophied parts of the epithelial layer. They do not suppurate; there is no bubo; they are not followed by a secondary stage. Spirochetes cannot be demonstrated.

TYPHOID FEVER.

When the history is incomplete, and before the rash appears, the two diseases resemble each other. The following, however, are the deciding factors: there is Widal reaction in typhoid, also nose bleed and diarrhea with abdominal symptoms; there is no leukocytosis; the rash of typhoid is raised, with roseola spots, and often occurs on the abdomen and chest; there is enlarged spleen; there is no alopecia; the Wassermann reaction is negative.

MALARIAL FEVER.

Malarial fever in the ordinary form, occurring in temperate climates, is evidenced by a chill and sweat; in the estivo-autumnal forms the fever is remittent. In all cases the plasmodium can be demonstrated; there is no leukocytosis; spirochetes are not present.

THE EXANTHEMATA.

Syphilis with fever, extreme sore throat, and a rash, might be mistaken for scarlet fever or measles, were it not for the following distinguishing symptoms: scarlet fever has no general adenitis in its first stage; the throat is red; there is no initial sore, no alopecia; spirochetes cannot be demonstrated. A strawberry tongue and desquamation occur at the end of a week or ten days.

RHEUMATIC FEVER.

Cases of syphilis with fever and severe arthritis might be diagnosed as rheumatic fever, but for the fact that in rheumatic fever there is no glandular enlargement. The arthritis is fleeting—from one joint to another—there is no rash, no alopecia; there is no primary sore. The Wassermann reaction is negative.

PSORIASIS.

Squamous syphilis might be mistaken for psoriasis; however the rash of psoriasis is distinctive. It is covered with small silverlike scales; there is no ulceration; it is chronic; it occurs on the extensor surfaces; as a rule there is no glandular enlargement, and no alopecia. Psoriasis rarely occurs on the face or hands.

CARCINOMA OF THE LIVER.

In syphilis, while the history of primary infection may be wanting, it is rarely absent; in cancer there is a previous history of indigestion and failing health, which is lacking in syphilis. There is no pain in the syphilitic tumor itself. Frequently, however, because of the local peritonitis surrounding a gumma, there is pain and frequently a friction fremitus can be both felt and heard. If the gumma be central the enlargement of the liver is general and cannot be distinguished from other cause of enlargement of the liver by its form, but if it is marginal, as it is apt to be, there is a huge tumor—often the size of a fist—frequently protruding in the epigastrium.

CIRRHOSIS OF LIVER.

The spleen is not enlarged as it is in certain forms of non-syphilitic cirrhoses. The liver is not hobnailed, but great sulci may occasionally be felt along the edges, due to contraction of fibrous tissue.

There are certain cases which resemble other cirrhoses most closely; these are enlargement of the liver, jaundice and fever; but here the diagnosis must be made by the Wassermann reaction, or by finding syphilitic lesions in other parts of the body, or by the therapeutic test.

A gumma of the liver in a beginning syphilitic lesion will disappear in a few days under inunctions of mercury. This will not be the case in any other liver lesion.

MULTIPLE NEURITIS.

Multiple neuritis might be mistaken for tabes dorsalis, but for the following features: there is no abnormality of the pupils; it is acute; there is no Wassermann reaction of the spinal fluid; there is no increase in the cells of the latter, and no increase in the globulin.

TUBERCULOSIS.

Syphilis of the trachea and the lungs is often mistaken for tuberculosis. In tuberculosis the tubercle bacilli can often be demonstrated; there is no Wassermann reaction; spirochetes cannot be demonstrated.

8. Diseases Due to Parasitic Infusoria

The following are some of the flagellatae, or organisms which at times —though rarely—find their habitat in certain organs of the body and give rise to disturbances of that particular organ.

Trichomonas vaginalis is common in acid vaginal discharges, and gives rise particularly to pruritus. Here the diagnosis from ordinary pruritus can only be made by examination of the vaginal discharge which will at once disclose the organism if the case is parasitic. Care of course must be taken to examine the urine, to be sure that there is no sugar present, because trichomonas can occur easily in cases of diabetes, although they are not at all common in these conditions.

Other organisms have their habitat in the intestines, and give rise to abdominal symptoms, such as pain and diarrhea of a chronic character. Dock's article describes *Trichomonas hominis* as the infecting organism in certain of these cases; in one of his cases, a hemorrhagic cystitis occurred.

Strong and Musgrave of the Philippines, believe certain *blastomyces* to be the cause in certain cases of chronic dysentery.

Other organisms are found in the expectoration, under various conditions, sometimes as a complicating organism, sometimes as the cause.

Diagnosis.—The only possible method of diagnosis in these cases is careful examination of the various excreta, so that one of the conditions caused by a parasite may not be looked upon as one of the ordinary cases of CYSTITIS, VAGINITIS or URETHRITIS.

D. Diseases due to Metazoan Parasites

1. Distomatosis

Organism.—Distomatosis is an infection by the *trematode worm.* These worms are flat, leaflike animals which deposit their eggs in the intestines or bronchial secretion. These eggs are extruded, find their way into an intermediary host—usually a mollusk; or they may become encysted on certain plants, the animal devouring the plant and encysted embryo. The worm finds its full development in the appropriate organ which it is wont to infest.

Clinical Forms.—According to Stiles:

The trematodes infest man from four different sets of organs:

(1) *Pulmonary distomatosis,* with cerebral or other infection as secondary. (2) *Hepatic distomatosis,* with splenic or intestinal infection as secondary. (3) An *intestinal distomatosis.* (4) A *renal distomatosis.* In addition there are rare instances of *ophthalmic distomatosis.*

PULMONARY DISTOMATOSIS.—Pulmonary distomatosis, lung flake disease, is the result of infection by *Paragonimus westermanii,* an oval pyriform fluke, 7 5/16 millimeters long, 4 to 8 millimeters broad, and 2 to 5 millimeters thick, with branched testicles and ovary and unbranched intestinal ceca. The eggs are yellow, 77 to 102.5 by 40 to 75 micrometers.

Development.—The eggs develop a ciliated miracidium in water after four to eight weeks and then enter a temporary host—probably a snail. They enter man in the infective stage, probably through drinking water. They are common in Japan (most of the work on these parasites has been done by Japanese), China and Formosa. The animals are found in cysts, in the lung and pleura especially but may be secondary in liver, abdominal cavity, brain, orbit, lower eyelid, cervical glands, and various other portions of the body. The parasite is found in the dog, tiger, and cat.

The *symptoms* of this disease are chronic cough with hemoptysis. The physical signs are extremely meager. The sputum contains myriads of eggs; it has much the appearance of pneumonic sputum. In extreme cases there may be signs of consolidation. The disease lasts for many years and may prove fatal; it apparently is incurable.

Differentiation.—This condition is constantly mistaken for TUBERCULOSIS.

Examination of the sputum will usually make the diagnosis, eggs being found and no tubercle bacilli. Besides this the extreme chronicity of the case with few physical signs and little fever makes the tuberculosis theory extremely doubtful, even without a sputum examination.

Cerebral Lesions.—Frequently individuals suffering from paragonimiasis are seized with convulsions epileptic in form and frequently jacksonian in type; they may develop a hemiplegia.

This condition may be suspected when it occurs in countries where pulmonary distomatosis is common. If the sputum of the individual contain eggs, then the diagnosis may fairly be made, for while an individual infested with distoma may have convulsions independent of the disease, the association is always suspicious of an animal having found a resting place in the brain.

The organism has been found in various other parts of the body, where it gives rise to symptoms referable to disturbance of the special organ involved. One rare case has been reported of Fasciola gigantica, but this was doubtless a case of chance parasitism, as the organism is common in Africa.

2. Teniasis

(*Tapeworm*)

Varieties.—The tapeworms most commonly affecting man are *Dibothriocephalus latus* (fish tapeworm) (Fig. 24), *Hymenolepis nana* (often with few symptoms) (Fig. 25), *Tenia saginata* (beef tapeworm) (Fig. 26), *Tenia solium* (pork tapeworm) (Fig. 27), and *larvae of Tenia echinococcus* of the dog (echinococcus disease).

Symptoms.—The commonest symptoms complained of by patients harboring tapeworms are indigestion, ravenous appetite, with weight, fullness and pain in the abdomen. Convulsions may occur in children and in individuals easily affected by any disturbing circumstance; anemia may be severe; white bodies are found in the stool; pruritus is common, especially nasal pruritus. Headache, vertigo and ringing in the ears occur. If these symptoms are the result of tapeworms, either the links of the

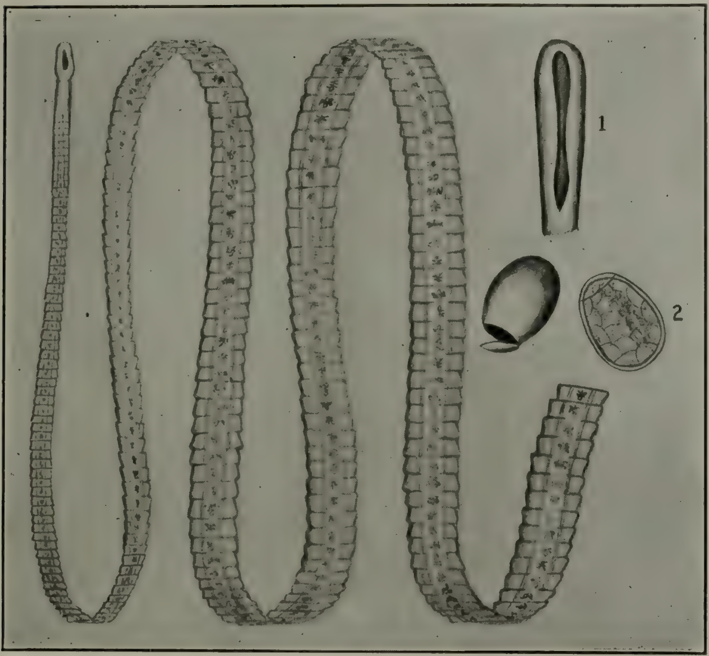

Fig. 24.—Figure of Dibothriocephalus latus. (1) Head; (2) Ovum. (After Eichhorst.)

worm or the ova can be found in the stool. These should always be demonstrated before a diagnosis is ventured (Figs. 24, 25, 26, 27, 28).

Conditions to be Differentiated from Teniasis

Pernicious Anemia.

The presence of a *grave anemia* should always lead to the examination of the feces for the worm or ova; finding these the variety can be fixed, and if the worm is the broad tapeworm or the fish worm, its removal will usually end the anemia.

There is no character of the blood of *essential pernicious anemia,* which may not be simulated by the secondary anemia due to the fish tapeworm.

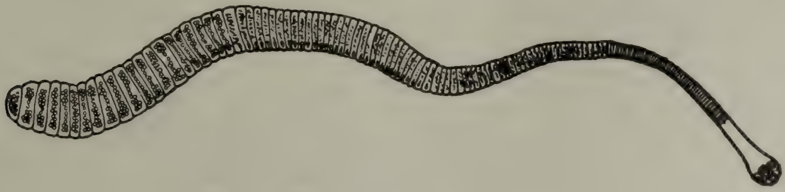

Fig. 25.—Hymenolepis nana. (Braun.)

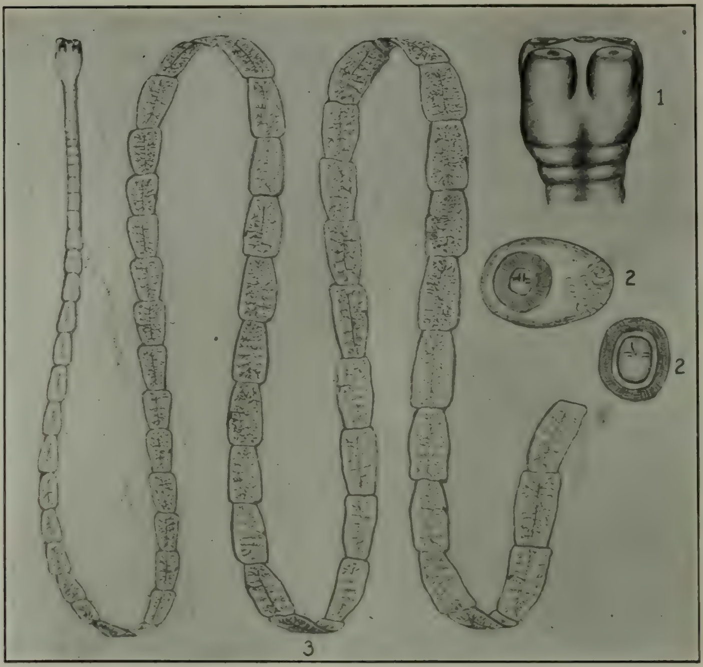

Fig. 26.—Tenia saginata—Beef Tapeworm. (1) Head; (2) Ova.
(After Eichhorst.)

The *diagnosis depends entirely upon the absence or presence of the worm, as demonstrated.* Beef tapeworms and pork tapeworms frequently give rise to convulsions and digestive disturbances which can also be differentiated by the presence or absence of the worms or the ova in the stool.

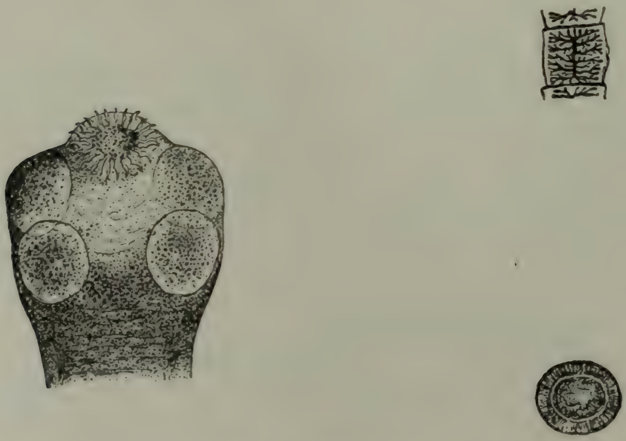

Fig. 27.—Tenia solium.

APPENDICITIS.

Appendicitis has been simulated. The author reported a case operated on for appendicitis, where three or four links of the beef tapeworm tænia saginata were found in the appendix. The case had all the local symptoms of appendicitis, but there was *no fever or leukocytosis.*

MUCOUS COLITIS.

Mucous colitis is a disease of the intestine in which the patient passes large pieces of mucus with stool. It is often taken for some form of tapeworm or for some other parasitic disease of the bowel. Examination of these pieces of mucus will prove their real character.

The Cysticerci

Local Symptoms.—The cysticerci of any of the tapeworms may give rise to local symptoms referable to the organ affected. One case reported by Osler had general soreness and stiffness of the muscles; there were nodules under the skin which proved to be cysticerci.

Osler also reports a case with symptoms of *diabetes,* which proved to have a cysticercus pressing on the floor of the fourth ventricle.

All varieties of nervous conditions have been simulated by cysticerci in various portions of the body.

When the ova become lodged in the *eye,* they may be diagnosed by the ophthalmoscope; such cases are on record.

Echinococcus Disease

Origin.—Echinococcus disease (larvae of *Tenia echinococcus*) is the result of contamination with the ova of the dog tapeworm and occurs most commonly in persons who live most intimately with dogs, and in dog fanciers.

Dimensions of Organism.—The adult worm is but 4 to 5 millimeters in length; hence its segments and even the entire worm may be overlooked in the dejecta of the dog, even though they be sought for.

Development.—Man becomes infested by digesting the egg and harboring the embryo which finds its way through the intestinal wall into the blood vessels, where it is carried to various organs—the liver perhaps being the most common site of lodgment.

The cyst is formed, and from the walls of this cyst others (daughter cysts) are formed, and from the walls of these, still others (granddaughter cysts); each is filled with a clear liquid containing hooklets.

Differentiation.—Echinococcus cysts of the liver give symptoms referable to that organ, and may be mistaken for:

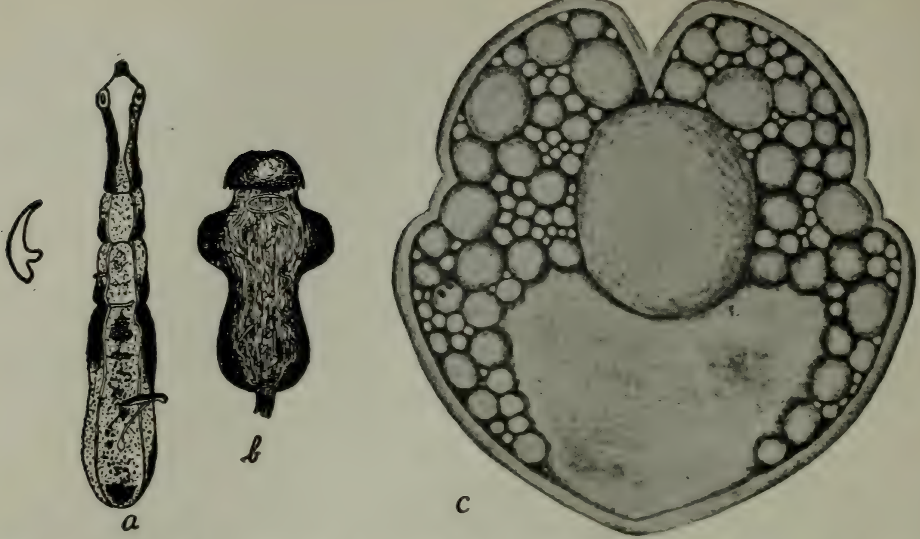

Fig. 28.—(a) Tenia echinococcus. Magnified 20 Diameters (Braun): (b) Diagram to Show Development of the Scolices of Tenia echinococcus and the Formation of Daughter Cysts; (c) Contents of an Echinococcus Cyst.

Cancer
Syphilis
Cirrhosis.
(For these see Echinococcus Disease of the Liver.)

Other organs give symptoms entirely referable to their disturbance or destruction. A differentiation from other states of the organs can only be made by tapping the cysts and finding hooklets.

3. Trichiniasis

Origin.—Trichiniasis, or trichinosis (the form of the word depending upon whether it is formed according to the Century Dictionary of *trichina* and *osis*, or according to Foster, of *trichina* and *iasis*), is a disease caused by the patient swallowing raw or partially cooked pork, containing the living embryos of the Trichina spiralis.

Organism.—Trichina spiralis is a nematode worm, viviparous, the male measuring 1.55 millimeters, having a double pointed caudal end, the female 3 millimeters in length, with a long pointed caudal end.

Development.—In this form the worm inhabits the intestine, giving rise to many embryos. These penetrate the intestinal wall and enter the muscle through the blood stream and the lymphatics, where they assume a spiral form and finally become encapsulated (Plate 2). It is this entrance of the embryos into the muscle which causes the mischief, giving rise to the symptom complex we call trichiniasis.

Symptoms.—The prominent features of trichiniasis are:

(1) Early edema appearing first in the eyelids. This may be so marked as to lead to a diagnosis of nephritis, the presence of albumin and casts in the urine giving color to this finding.

(2) Pain and swelling of the muscles with joint pains and edema of the skin over the muscles. The pain and stiffness of the limbs is the chief complaint of the patient, so great is the swelling of the muscles that they are hard and tense. The swelling and tenderness of the masseter, the muscle standing out as a mass just anterior to the ear, sometimes causes a diagnosis of mumps. The muscle, however, can easily be isolated by grasping it inside the cheek and having the patient close his mouth.

(3) Fever.—In cases of trichiniasis in which a number of individuals in one locality are simultaneously seized with muscle pains and fever, trichiniasis should at once be suspected, especially if the affected individuals are, as suggested by Osler, Germans who have participated in a "Fest," but when an isolated case occurs, and the individual develops a continued fever, the diagnosis is almost without exception erroneously Typhoid Fever.

The fever of trichiniasis is usually prolonged for three or four weeks and is remittent or intermittent, but it may be in some cases of a continued type resembling chiefly the fever of typhoid. It is only after careful examination of the case—including a blood examination—that a correct diagnosis can be reached.

(4) The urine is often excessive in amount, averaging sixty ounces in twenty-four hours. It contains a trace of albumin and many small dark granular casts; the latter slowly disappear during convalescence.

(5) Gastro-intestinal Symptoms.—In many cases no such signs can be developed except by careful questioning. There is usually a history of eating partly cooked meat, but this is often forgotten by the patient. The gastro-intestinal symptoms, when present, vary from a severe gastro-enteritis to a mere griping pain. Worms have been found in the stools; they are not found in the majority of cases.

(6) The Blood.—Thomas K. Brown in examining the blood of a patient in a case of supposed typhoid fever, noticed first that there was a marked leukocytosis, and in a differential count that the eosinophils were greatly in excess of the normal, about 23 per cent. Subsequent observation on this same case showed that the leukocytosis and the eosinophilia persisted for several weeks. Observation of five cases showed leukocytosis and eosinophilia in all. These cases were reviewed by Osler (Am. J. of Med., series for 1899, vol. 1, page 251). The blood in one case reported by the writer was as follows:

Date	*Red*	*White*	*Hemoglobin* %	*Polymorphonuclear Cells*	*Eosinophils*	*Lymphocytes*	
						Large	*Small*
11- 6-06	4,580,000	23,800	..	69	23	2	6
11- 7-06	4,649,000	21,200	75	58	39	..	..
11- 8-06	....	13,000	..	60	27	8	5
11- 9-06	4,800,000	12,600	80	41	27	24	8
11-10-06	....	10,200	..	53	22	15	10
11-11-06	....	9,500	..	57	29½	9½	..
11-12-06	4,420,000	9,000	..	69	20	6	5
11-14-06	....	12,400	..	61	33	..	..
11-16-06	....	11,650	..	55	27	..	..
11-18-06	4,350,000	9,600	75	57	25	14	4
11-20-06	....	7,800	..	48	20	..	..
11-26-06	4,200,000	9,600	70	..	22	..	..
12- 5-06	....	12,000	..	..	..	..	..

Conditions to be Differentiated from Trichiniasis

There is little difficulty in making a diagnosis of trichiniasis when there is an epidemic; the greatest difficulty exists when cases are isolated, as so often happens. It can be confused with:

Typhoid Fever (in isolated cases)
Rheumatism
Corrosive Poisoning
Mumps.

Typhoid Fever.

Trichiniasis resembles typhoid fever in many respects; the patient slowly begins to ail; a fever arises; the individual complains of pain in various portions of his body, without special localization. Sometimes there is diarrhea; in severe cases there is delirium—so that the picture of typhoid fever is well simulated.

The presence of trichiniasis may be suspected if the patient has lately eaten undercooked pork. Examination of the *blood* will determine the diagnosis at once, for, as has been stated, there is a leukocytosis and eosinophilia accompanied by continued fever almost without exception in trichiniasis; this is never present in typhoid fever. The Widal reaction and typhoid bacilli can be demonstrated in typhoid fever.

Rheumatism.

In rheumatism there is an arthritis. In trichiniasis the pains which are so common are in the muscles in the length of the limb or over the trunk; these parts are swollen and tender. The arthritis of rheumatism quickly affects one joint after another.

Corrosive Poisoning.

The occurrence of diarrhea in certain cases which persist for a long time may give color to the thought that the case is one of corrosive poisoning, but the fever, the leukocytosis, the eosinophilia, and the painful muscles are absent in corrosive poisoning, while diarrhea is a prominent symptom.

MUMPS.

Trichiniasis is sometimes mistaken for parotitis. I have seen the mistake made, due to the fact that there may be swelling and tenderness of the masseter muscle, giving the face a prominent appearance in that position, and making a picture not unlike mumps. Indeed, in one of the cases reported by Dr. Mellersh and myself that diagnosis was made by the interne.

4. Ascariasis

(Round Worm)

Organism.—Ascariasis is a condition due to an infection by the Ascaris lumbricoides. This worm finds its lodgment in the intestinal canal.

Symptoms.—The symptoms which accompany it are meager. Frequently in children there are symptoms of indigestion; there may be convulsions, there may be nervous symptoms, but these are so common in connection with other diseases, that their mere presence must not be taken as indicating an infection with the round worm.

Diagnosis. — The diagnosis can be made only by finding either an adult worm in the stools, in the vomitus of the patient, or by finding the characteristic ova in the stools (Fig. 29).

Mucous colitis by reason of the threads of mucus extruded from the bowels, is very frequently mistaken by the laity for round worms. Examination of the stool will of course at once make the diagnosis.

Epileptiform reflex convulsions due to other causes may be differentiated by clearing the in-

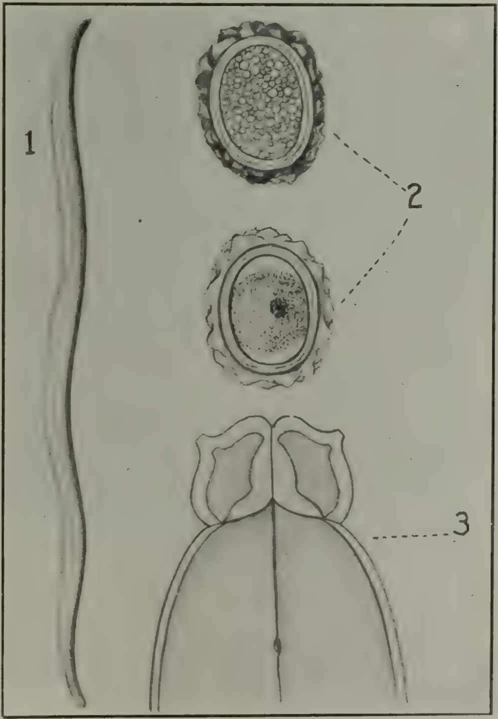

Fig. 29.—Ascaris lumbricoides. (1) Adult Worms; (2) Ova; (3) Head. (From Eichhorst.)

testine of worms. If the convulsions continue after entirely ridding the intestinal tract of worms, the worms were not the cause of the convulsions.

5. Uncinariasis

Origin.—Uncinariasis, variously known as *ankylostomiasis, hookworm disease, St. Gothard's tunnel disease, Egyptian anemia* and *brickmaker's anemia,* is due to the presence in the intestine of man, of a parasite—a small round worm, one of the varieties of the *Uncinaria.* Two species are responsible for the disease—the old world hookworm and the new world hookworm — the symptoms of the disease caused by the two species being identical. It probably gains entrance to the system by means of the skin, ground itch being the first symptom.

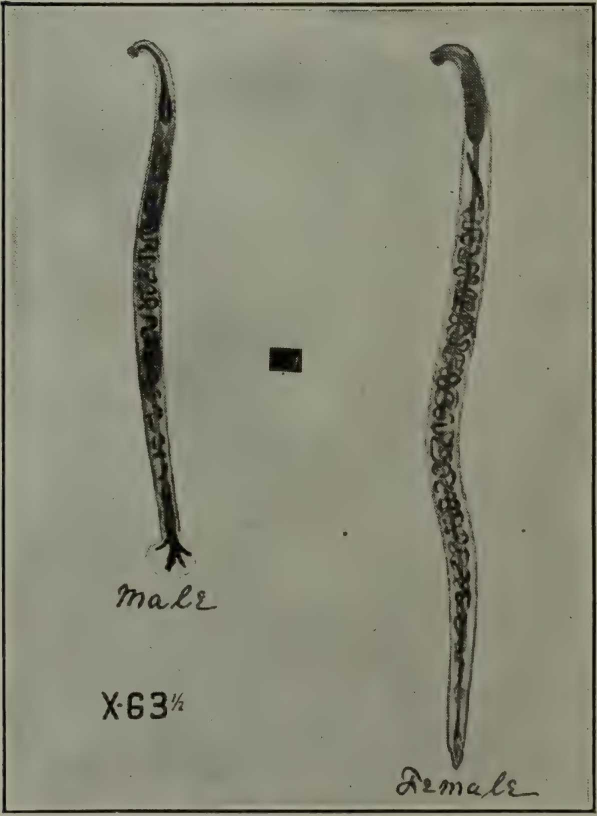

Fig. 30.—Adult Necator americana. (Personal Observation.)

Varieties. — The worm most common in this country and native to it is the *Necator americanus* (new world hookworm). The other variety is *Ankylostoma duodenale* (old world hookworm).

Necator americanus.—Until Allen J. Smith and Charles W. Stiles made their brilliant observations, the disease which was then usually called *ankylostomiasis* was looked upon in this country as a mere curiosity not likely to be encountered here. Since Stiles discovered the species of the worm *Uncinaria americana* to be a native of the entire southern belt of this country, and that the great mass of the rural population of the South is infested by it, the disease has become a real entity—a serious problem. Constantly cases are cropping up in the northern temperate zone of the country.

The worm (Fig. 30) *Necator americanus* (new world worm) is from 7 to 11 millimeters (about $\frac{1}{3}$ inch) long, and less than 1 millimeter (1/25 inch) in diameter. It is hooked at the head end, the curve at the head giving the common name *hookworm* to the parasite. At the mouth it has a pair of central lips; it has four lancets and a prominent dorsal tooth—five in all (Fig. 31).

The vulva or sexual opening is in the anterior half of the body of the female, which is slightly larger than the male. The male has a series of caudal rays (Fig. 32) in the bursa with which it seizes the female in copulation, the two worms being at right angles and firmly fixed. Ova are 64 to 72 micrometers in length, 36 to 40 micrometers in width, oval and somewhat blunt at the ends, and often show cellular segmentation. Their size, therefore, is about eight times the size of a red blood corpuscle (Fig. 33).

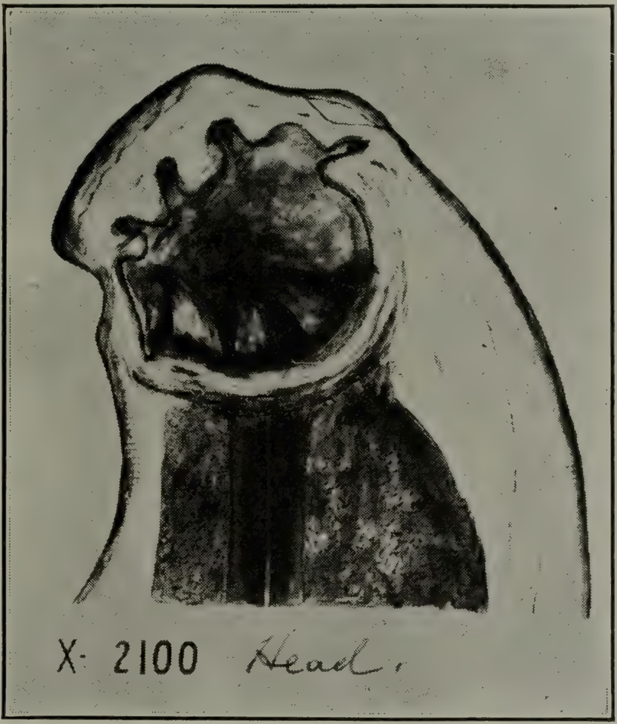

Fig. 31.—Head of Necator americana. (Personal Observation.)

Ankylostoma duodenale.—Ankylostoma duodenale cannot be differentiated from new world worm by the naked eye. It is from 8 millimeters (1/3 inch) to 18 millimeters (3/4 inch) in length. It has two pairs of cervical teeth and two knoblike enlargements dorsally. The eggs

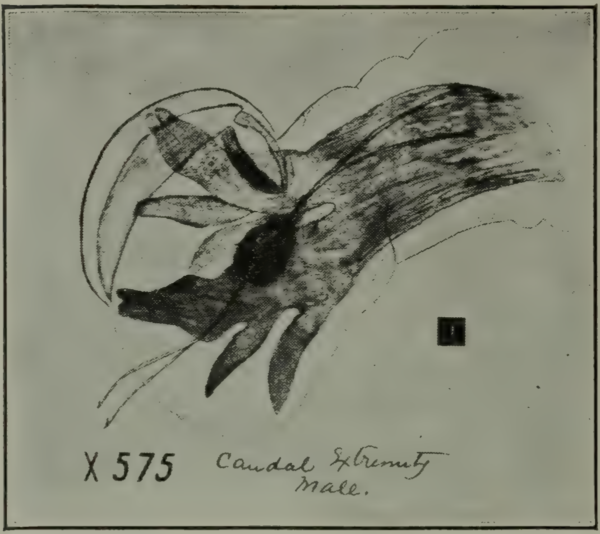

Fig. 32.—Necator americana. (Original Observation.)

are from 52 to 61 micrometers long, and from 32 to 38 micrometers broad, decidedly round at the ends. The sexual opening is behind the middle point of the worm.

Geographical Distribution.—Hookworm disease is extremely common in the Southern United States and Porto Rico, 90 per cent of the inhabitants of Southern Florida and Porto Rico being infested, according to

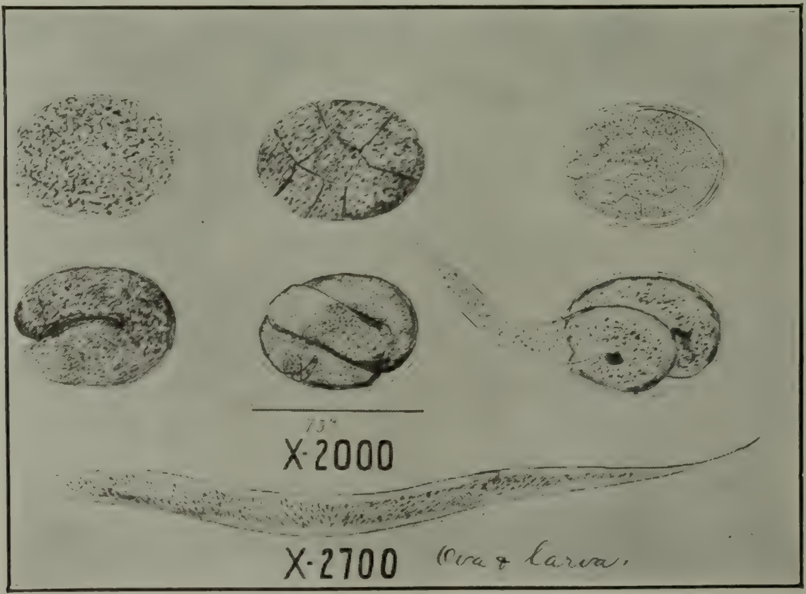

Fig. 33.—Ova and Larva of Necator americana. (Original Observation.)

Stiles, Ashford, King and others. Thirty to eighty per cent of the German miners are said to be affected. Miners in the United States district are fairly free from the disease.

Symptoms.—*Blood.*—It apparently makes but little difference how slight the infection, the case always shows some anemia. Some cases which have but a few worms, have 70 per cent hemoglobin, while the more severe cases have a much lower hemoglobin. There is a corresponding reduction in the number of red cells, and there is usually slight leukocytosis.

Ashford and King believe that aside from the differential count the hemoglobin examination is the most important of the blood examinations. Practically all of their cases show lack of hemoglobin. The leukocytes were not above normal in any of one hundred cases.

The differential count is important because it shows the most important characteristic sign of the blood, namely eosinophilia. In the one hundred cases reported, practically all showed this. In regard to this sign the authors concluded that (1) uncinariasis is marked in some part of its course by eosinophilia, which is greater in the acute than in the chronic cases, and (2) an increase of eosinophilia under treatment is a good prognostic sign. The red cells are usually reduced, one count in the Porto Rico series being as low as 904,000.

Stools.—These invariably contain the ova of the worm. After a purge the adult worm can be found.

Fever.—In a few cases there is a rise of temperature.

Skin.—The skin is dull, pasty white, varying in appearance. When accompanied by the lowered mentality as it is in some cases, they resemble myxedema. The skin is dry and harsh. The hair is also dry and harsh. There is but little hair in the arm pits and genitals of our case. In about 5 per cent of the Porto Rico cases there was papular eruption on the legs—ground itch.

Nervous Symptoms.—The face indicates the apathy of the individual; this was marked in our Case 2. He always looked tired and spoke with a drawl; he would sit down immediately when not forced to stand; vertigo, headache, diminished sexual powers, signs of peripheral neuritis and of cord involvement were present. This was doubtless also the condition in our Case 1.

Circulation.—The heart is almost always increased in size, as in Case 2. Anemic murmurs are present, and edema, palpitation and dizziness are common symptoms. The increased area of the heart dullness is about the only one of the symptoms that cannot be accounted for by the anemia. It would appear that either the heart muscle itself, or the nervous supply is so affected that the heart dilates more than it would with ordinary anemia.

Digestive Symptoms.—Our first case shows the common symptom of epigastric distress. It will be noted that this was present in this apparently milder case, while it was absent in the more severe one. Hemorrhages in the stools were never noted in the Porto Rico cases. Neither in our cases, nor in those of any of the others is there a report of the presence of occult blood.

The appetite is precarious, many of the patients being "dirt eaters." One writer believes that the eating of dirt causes relief of the epigastric symptoms.

The patient is always underdeveloped. This has been shown to be the fact in the carpal development so brilliantly shown by Rotch. The peculiar apathy of these patients has led to the popular application of "lazy" to the worm and the cognomen of "lazyworm disease" to the malady.

Diagnosis.—The diagnosis of this disease depends absolutely upon the use of laboratory methods by the practitioner. It is true that an expert could pick out from a company of individuals those seriously affected, but the *examination of the blood and stools is necessary to confirm the diagnosis.*

As I have pointed out in other places, the use of the microscope is necessary for all of us, not only in this condition, but in many others, if we are to hold our own in the work of medicine with the young men who are constantly coming in. In uncinariasis a hemoglobin estimation will show loss of coloring matter. This can be done quickly with a Tallqvist scale. Counting of the corpuscles will show a diminished number of red cells, and an approximately normal number of white ones. A differential count is made easily and quickly by making a smear and staining with Wright's stain. This differential count will show the eosinophilia spoken of above.

Examination of the stools is easily done. A bit of the feces is spread on a slide with a drop of water and quickly examined under a microscope under low power. These two processes are the only essentials in making the diagnosis; without them—at least without the latter—it is impossible.

Given a case of anemia with eosinophilia, without marked leukocytosis, and occurring in the subtropical or tropical regions, the chances are that it is a case of uncinariasis. The examination of the stools will confirm or set aside the diagnosis. In examination of the stools the ova of other worms will be found, such as the round-worm, the pin-worm and the whipworm.

Conditions to be Differentiated from Uncinariasis

The conditions can be mistaken for:

Pernicious Anemia
Chlorosis
Myxedema
Other Intestinal Worms
Malarial Fever.

PERNICIOUS ANEMIA.

This occurs as a well-developed disease in individuals over thirty years of age in the great majority of cases. Uncinariasis attacks individuals of all ages. Examination of the blood will show a marked eosinophilia in uncinariasis, but *not* in pernicious anemia. Otherwise the blood of pernicious anemia may closely resemble that of some cases of uncinariasis. There is an absence of ova or of parasites in the stools of pernicious anemia.

CHLOROSIS.

Chlorosis occurs exclusively in young girls. The hemoglobin is much reduced, the reds but little; there is no eosinophilia, no ova in the stools and no parasites.

MYXEDEMA.

Myxedema is not characterized by anemia; there is no eosinophilia; there are no ova. The dull intellect is at once cleared up by the use of thyroid extract; this has no effect on uncinariasis. The cases abound in every region.

Other intestinal parasites can be differentiated only by familiarity with the worm and its ova.

MALARIAL FEVER.

Malarial fever, on account of its anemia, might be mistaken for uncinariasis by the careless observer, but the intermittent chills and fever characteristic of this disease are not seen in uncinariasis; added to this the presence of malarial organisms and absence of eosinophilia will make the case a certainty.

6. Filariasis

Organism.—This condition is due to infection of the human being by the worms of the genus *Filaria.* The adults of the species inhabit the lymphatic and connective tissues of man, and their embryos are found in the peripheral blood. There are two of these filaria which are of importance from the medical standpoint. One is the *Filaria sanguina hominus or Filaria Bancrofti,* and the other *Filaria loa.* According to Stiles there are many species of this genus, but so far as is known at present, these two are the only ones that are of any importance in human medicine.

Mode of Infection.—The disease is spread by the bite of mosquitoes; this fact was discovered by Manson many years ago. When a mosquito bites an individual who is infected with filaria, the larvae are taken into the blood. They then undergo development in the muscles of the mosquito and there reach a stage in which they can be transmitted to man by its bite.

Filaria Bancrofti.—Filaria Bancrofti is a worm, whitish or brownish in color, from 95 mm. to 1 cm. in length, and .26 mm. in diameter; it is viviparous. The larvae are from 300 to 340 μ long; they are very much more numerous in the peripheral blood during the night than during the day time; they may be seen in a specimen of blood under a cover-glass, swimming freely among the corpuscles and whipping them about by the vermicular motion (Fig. 34).

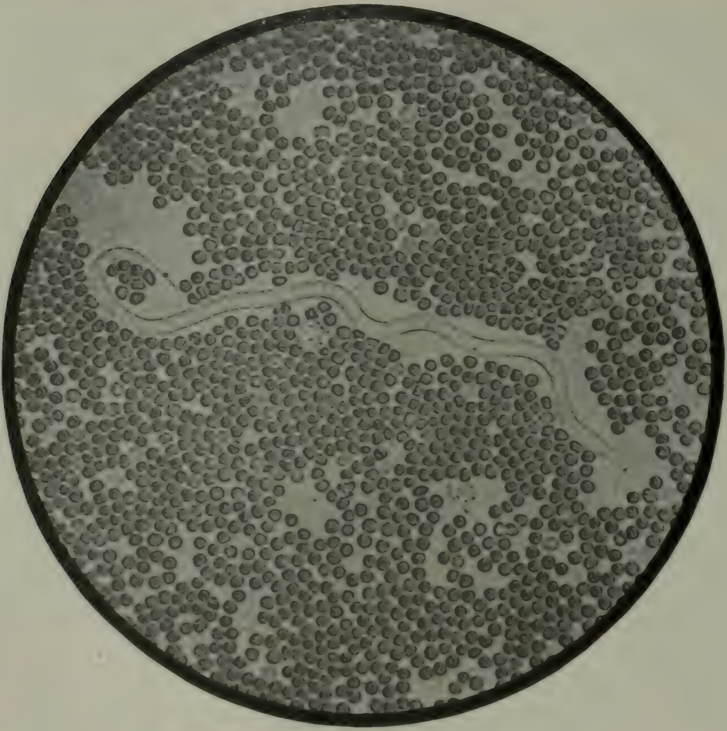

Fig. 34.—Embryo of Filaria in Blood.
(From Bull. No. 1, 1913, U. S. A. Reports.)

Symptoms.—The adult worm, by finding lodgment in the lymphatics, gives rise to various symptoms—abscesses, lymphangitis, elephantiasis, enlarged varicose glands, chyluria and chylous edema of various portions of the body, legs, scrotum, etc.

The attention of the physicians in the tropics, where this worm is common, is usually attracted first by either a lymphangitis, enlarged varicose glands, elephantiasis or chyluria. Occasionally without any impairment of the health, an individual suddenly passes chylous urine, sometimes tinged red. The passage of the urine lasts but a few days at a time and may entirely disappear. Usually if the blood be examined at night the larvae may be found circulating freely in the blood.

Lymphangitis is usually accompanied by fever which may rise as high as 105° F. Repeated attacks of this lymphangitis in the scrotum and in the legs give rise to elephantiasis. In elephantiasis the affected parts are often of huge size; the skin is thick, creased and rough—creased in huge folds. Filaria are not found in every case of elephantiasis.

Diagnosis.—In all cases of chylous urine, lymphangitis, varicose glands and elephantiasis occurring in the tropics, the blood should be carefully examined for the presence of larvae in the blood. Being found, the diagnosis is certain and can be mistaken for nothing else.

Filaria loa.—The Filaria loa lodges in the connective tissue of the body and gives rise to annoying, rather than dangerous symptoms. Occasionally it appears near the surface, in the eyelids, and on the bridge of the nose where it can be seen under the skin and can there be removed.

The larvae of this worm can be found in the blood during the day time; they disappear at night.

Symptoms.—The chief symptoms caused by this worm are itching and creeping sensations, with edematous swelling in different parts of the body—so called "calabar" swelling. It suddenly appears in various portions of the body, moving slowly along, and in about three days entirely disappears; it is painless and slightly irritating.

Diagnosis.—Here the diagnosis is made at once by examination of the blood. However, it is stated that morphologically the larvae of the Bancrofti and the Loa are indistinguishable. The diagnosis can be made by the general symptoms presented by the patient, and by the fact that the *larvae of Loa appear in the day time, while the larvae of the Bancrofti appear at night.* The former are probably transmitted just as are the worms of the Bancrofti.

This disease can always be distinguished from others under consideration by examination of the blood for larvae.

Conditions to be Differentiated from Filariasis

Malaria
Non-parasitic chylous urine
Elephantiasis
Lymphangitis
Hernia.

MALARIA.

Malaria is frequently a diagnosis when there is lymphangitis in a case of filaria, but the fever of malaria runs its regular course, and the blood shows the germ of malaria fever.

NON-PARASITIC CHYLOUS URINE, ELEPHANTIASIS, OR LYMPHANGITIS, ETC.

These conditions are not very likely to be mistaken in temperate climates. Positive diagnosis can only be made by examination of the blood.

HERNIA.

Hernia may simulate a lymph scrotum, but careful examination and the finding of an open ring will be certain to clear up the diagnosis. Embryos are not found in the blood.

7. Dracontiasis

(*Guinea-worm Disease*)

Organism.—This is an infection by *Filaria medinensis.* The parasite is found in the leg near the ankle. It does not remain here, however, but wanders through the other portions of the body and curls in some spot under the skin; the swelling increases, becomes vesicular, and breaks down into an ulcer. From this ulcer the worm slowly protrudes its head.

Geographical Distribution.—Occasionally it occurs in the United States. It is common in the East Indies and in Africa.

8. Oxyuriasis

(*Thread-worm*)

Organism.—This condition is due to the lodgment of the thread-worm, or *Oxyuris vermicularis,* in the intestinal canal. It inhabits the great bowel. The term "seat-worm," which applies to this parasite, originates from the fact that they so constantly make their appearance at the anus. However, they may occur anywhere in the intestinal canal.

Symptoms.—The writer has seen the appendix packed with these worms, giving the symptoms of acute appendicitis. They are constantly the cause of itching about the anus in children and in adults. Occasionally they cause itching about the vulva by reason of the fact that they have escaped from the anus, and reaching the vulva cause a marked irritation; such symptoms as these are frequently mistaken for eczema, and, vice versa, simple eczema is frequently thought to be due to the thread-worm.

General symptoms rarely occur. Convulsions are such a common result of other conditions, and so rarely the result of parasites, that all other conditions must be excluded before the presence of worms is taken as the cause.

Diagnosis.—This may be made absolute by examination, whereas if oxyuris is actually present it may be seen about the anus or in the vulva.

Purgation will cause a discharge of both worm and ova; they may be seen in the stools, the worms moving about like live threads.

The parasite is a small, round, white object measuring 3 to 5 mm. for the male and 10 mm. for the female, and from .16 to 6/10 mm. in diameter (Fig. 35).

Conditions to be Differentiated from Oxyuriasis

The condition may be mistaken for:

Anal or vulvar itching, from any cause
Diabetes mellitus
Eczema
Itching of the nose
Appendicitis.

Anal of Vulvar Itching.

Local examination should always be undertaken to differentiate the causes of itching of the genitals or anus. The worms and ova must be sought for on the skin and in the feces. Failure to find them will of course make negative a diagnosis of oxyuriasis.

Diabetes mellitus.

The diagnosis of diabetes mellitus can be substantiated by examination of the urine and finding of sugar. Itching of the genitalia is the symptom which gives rise to confusion.

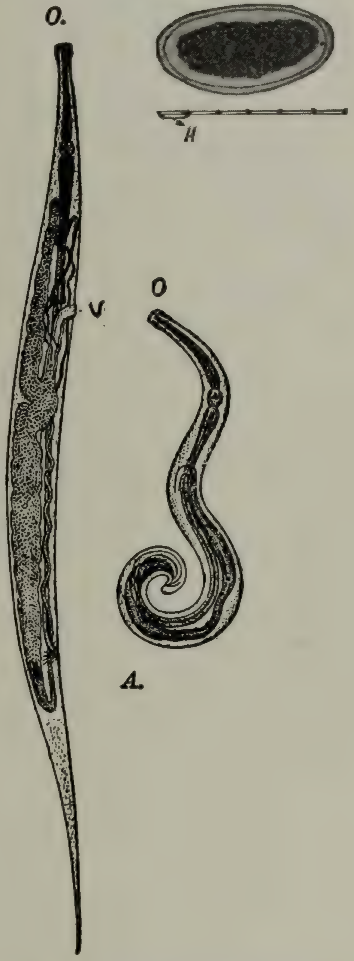

Fig. 35.—Oxyuris vermicularis: to the Left, Female; to Right, Male (Considerably Enlarged). A, Anus; O, Mouth; v, Vulva; u, Ovum of Oxyuris vermicularis. (Braun, after Claus.)

Eczema.

Eczema may be the result of irritation by these worms, or it may be complicated by them. Getting rid of the worms will cause the eczema to recover if the irritation is due to their presence.

Itching of the Nose.

Itching of the nose, giving rise to picking of that organ, is not a symptom of intestinal parasites except as the result of the indigestion which they cause. As a rule parasites are not present in the feces of children with the habit of nose picking. The seeds of bananas have been frequently mistaken for pin-worms, and they look not unlike them; however, they are not living when passed; a microscopic examination will reveal this difference.

9. Diseases From Arachnoids and Ticks

Organism.—*Linguatula rhinaria* is a pentastoma, which has its habitat in the nasal cavities of dogs, wolves and horses. When it infests man, it gives rise to severe nasal symptoms, and is found usually in the larval state.

Diagnosis.—The diagnosis can only be made by microscopic examination of the nasal secretion, and discovery of the ova or larvae themselves.

10. Scabies

Organism.—This troublesome skin disease is caused by the *Sarcoptes scabiei.* The female of this minute animal, or itch mite, burrows in the skin, making a furrow about 1 cm. long. The animal may be found at the end of this furrow.

Site of Infection.—It is most common between the fingers and in the nipples of women. Vesicles appear in the region of the burrow, but the excoriations and widespread eruption are caused by the scratching of the patient. The disease is always very contagious and frequently spreads throughout the family of the infected individual.

Diagnosis.—The disease may be mistaken for eczema, and for the scratch marks common while individuals are infested with body lice.

The diagnosis of scabies can always be made by *observing the peculiar burrows made by the female, and also positively by finding the animal at the end of the burrow.* The entire body is usually covered by scratch marks. If the itching is DUE TO THE ACARUS, the marks and eruption are in the folds of the skin, in the elbows, under the arms, about the genitals, etc. Eczema is general over the body, and rather on the external surfaces of the arms and legs. There are no burrows or galleries resembling those made by the itch mite.

11. Pediculosis

Organism.—Pediculosis is due to the Pediculus capitus, Pediculus corporis, or Pediculus (Phthirius) pubis, which affects the head, trunk or pubes.

The insect is minute. It rapidly fills the hair of the head, arm pits, or pubis with nits—minute whitish bodies fastened to the hair.

Symptoms.—The symptoms are itching of the body with scratching marks—the result of the victim's attempt to relieve himself. Occasionally there are present the tache bleuâtres, round, bluish spots, covering the abdomen and thighs. There may be eczema, and sometimes fever.

Diagnosis.—The diagnosis can be made by observation of the insect itself or the nests of eggs tightly adherent to the hair.

Conditions to be Differentiated from Pediculosis

It may be mistaken for:

Scabies
Itching of general pruritis
Jaundice
Typhus fever.

SCABIES—ITCHING OF GENERAL PRURITIS—JAUNDICE.

The absence of insects or eggs in their cases adherent to the hair will at once make a negative diagnosis.

TYPHUS FEVER.

Typhus fever might be mistaken in the febrile cases of pediculosis with the bluish petechial spots. Now that it is known that cases of typhus practically always have pediculosis, the differentiation may cause some trouble, but the high fever of typhus, the typical rash on the third or fourth day and the presence of other cases of typhus, will help in the diagnosis.

12. Myiasis

(*Myiosis*)

Species.—Various species of different genera of flies have found lodgment in or on the bodies of men, and give rise to symptoms referable to disturbances of the special organ which the fly has infected.

The screw-worm has very frequently been found in the nose and in wounds—it is the larva of *Lucila macellaria.* It gives rise to local inflammatory conditions.

Frequently when there is suppuration in the ear, eye or conjunctiva, the blue-bottle fly larvae have been deposited. Swan has reported the larvae of the common house fly, *musca domestica,* in the ear.

The latter fly also gives rise to a gastro-intestinal myiasis. Osler cites various other flies whose larvae are found in the intestinal tract.

Symptoms.—The symptoms which arise are those of a disturbance at the seat of infection: when the intestine is affected there is more or less severe gastro-intestinal disturbance. Coyane has reported two deaths.

The importance of this condition should not be underrated, notwithstanding the rarity. Certain hysterical individuals believe they have these larvae in their stomachs and intestines; before being sure that such conditions are not present, a careful microscopic examination should be made of the stomach contents and stool to be certain of the absence of larvae. It can be distinguished from any other local condition by the discovery of the larvae of flies in the discharges.

13. Systemic Blastomycosis

The facts in this chapter are taken from an article by A. N. Stober in Archives of Internal Medicine, April, 1914, the report of the cases being those in and about Chicago.

Etiology.—This disease occurs in early life. The occupation of the individuals is almost always one involving manual labor. The majority of the cases were foreigners, who were poor. The cause has been proven, according to this author, to be due to a mold.

Organism.—The organism has been recovered from the blood; it is present in the pus of unopened abscesses, in the sputum, urine, and feces. Experimentation on animals demonstrated its pathogenicity. The organism was found in the blood of one patient.

Symptoms.—Most of the patients cited began their illness with an acute infection of the respiratory tract. There was pain in the chest, fever, dyspnea, cough, expectoration of bloody mucus. After the establishment of the disease, there was loss of strength, emaciation of the part affected, irregular fever, rapid pulse. The joints, when they were affected, were very much enlarged and the temperature was irregular. In almost all cases symptoms of pulmonary infection occurred; the sputum was thick and purulent, and often contained blood; cough was present, sometimes severe.

Blastomyces were present in the sputum in eight cases out of the series. Areas of consolidation occurred first, as a rule in the apices.

Differential Diagnosis.—The differential diagnosis is thus summed up by Stober.

"The conditions with which blastomycosis has most often been confounded and the important differential points will be briefly enumerated.

Coccidioidal Granuloma.—This disease has been ably discussed in the articles by L. Hektoen and O. S. Ormsby. That the disease from a clinical and pathological point of view closely resembles blastomycosis is generally conceded. The principal differential points advanced by the various authors are that in coccidioidal granuloma the nodular lesions resemble more closely those of tuberculosis; there is a greater tendency to involvement of the lymph-nodes, the cutaneous lesions are more ulcerative, the average duration of the disease is shorter than in blastomycosis, and reproduction of the organism in the tissues occurs principally by endosporulation.

Epithelioma.—The cutaneous lesions in one case and also in other cases were first regarded as epithelioma, but close observation should readily differentiate these conditions. The principal points are the slower growth of epithelioma, the greater induration—extending into the tissues beyond the ulcer margin, and the absence of the bluish-red halo surrounding the border, which contains the characteristic miliary abscesses, and in which the blastomycotic organisms are usually found.

Tuberculosis.—Reference has already been made to the close resemblance between blastomycosis and tuberculosis, both clinically and pathologically. So far as the symptoms of the pulmonary infection are concerned, the clinical pictures of the two diseases are so similar that differential diagnosis is extremely difficult. Tuberculosis is the more destructive of the two, and cavity formation and marked hemoptysis are therefore more common. The examination of the sputum in most cases of course gives positive evidence of the condition present, and blastomycetes should be looked for in those cases of suspected tuberculosis in which the tubercle bacilli are persistently absent.

The affections of the bones are also very similar. The small bones and joints are perhaps more often affected in blastomycosis, the lesions are more often multiple, and associated pulmonary, cutaneous and subcutaneous lesions are more common. It should be noted that in several cases the blastomycotic ulcerations of the skin were mistaken for lupus. A very interesting and important point emphasized in Case 23 was the marked simulation of Pott's disease by the blastomycotic lesion of the spine. If any differences exist it might be stated that blastomycosis is more painful and rapid, and may begin by extension from the pleurae or adjacent structures rather than in the bodies of the vertebrae.

The tuberculin reaction was tried in several of the cases in this series, with negative results.

Syphilis.—Some of the pustules and ulcerations might be mistaken for syphilitic lesions and the hard indurated character, with subsequent softening of the deep abscesses, is suggestive of gummata. Röntgenograms of the affected bones have shown osteoporosis and periosteal thickening almost identical to that produced in syphilis, and one might be further mistaken and misled by the improvement which follows the administration of potassium iodid.

Inasmuch as positive diagnosis of obscure cases of syphilis are now being made with the Wassermann serum test, and it being realized that this is a group reaction, it was deemed of interest to try the reaction with blastomycosis. The serum in two cases was tested by Dr. F. G. Harris. One proved negative and the other was positive. It was assumed that the latter patient was at one time affected with syphilis, although no history could be obtained. The blastomycetic vaccine reaction may prove to be a positive factor in the diagnosis of blastomycosis. In three cases of blastomycosis in which cutaneous lesions were present, a distinct inflammatory reaction was observed in the local lesions after the injection of the blastomycetic vaccine filtrate. In the two cases which were not so far advanced a rise of temperature was obtained in from six to twenty-four hours after injection, the temperature in some instances persisting for two or three days. Such a rise in temperature did not occur in two cases of syphilis, three cases of tuberculosis and three normal persons who were subjected to the test.

Cutaneous tests consisting of the application of the filtrate and also triturated cultures in lanolin to scarified areas provoked no reaction sufficiently distinct to be considered specific. Ophthalmic tests with the filtrate were also negative. Different results might have been obtained with the use of cultures killed with less heat or in some other manner inasmuch as the ones used were subjected to a temperature of 110° C.

Dr. Boughton, using suspensions prepared by Dr. L. Hektoen, noted the formation of small nodules at the site of puncture and inoculation after the method of von Pirquet. These did not occur in a subsequent test. Other cutaneous and ophthalmic tests with these suspensions were negative."

14. Bacillus ærogenes Infection

(Fulminating Gangrene)

Etiology.—Any one having seen this infection can scarcely mistake it for any other condition. There is always the history of injury; frequently the latter is distinctive—a crush, sometimes a compound fracture or a fracture which appears simple—but there is in fact a slight communication between the skin and the injured area. The author has seen it from a hypodermic injection in a leg, the seat of arterial thrombosis. Gwynn reported a case following a hypodermocylsis.

Symptoms.—The first symptom is usually fever, then rapid discoloration, swelling and emphysema of the parts, the whole of the tissue becoming discolored, edematous and ill-smelling and the seat of gangrenous emphysema. At first there may be dark—almost black—pus and the kind of infection is not suspected. Sometimes the part takes on the appearance of erysipelas, but the edges of the inflammation are not raised: the color is dark brown. Blebs rapidly occur with blue-greenish appearance; the exudate in these blebs is of foul-smelling liquid of sweetish odor. In typical cases pain is absent. The process extends by direct continuity as the gas dissects the parts.

Conditions to be Differentiated from Bacillus ærogenes Infection

The condition must be distinguished from:

Erysipelas
Streptococcus or Staphylococcus infection
Gangrene due to destruction or obliteration of the vessels.

From all these is the marked illness of the patients, all such individuals seem at once dangerously ill.

ERYSIPELAS.

Erysipelas has a hard red infiltration of the skin with raised edges. There are blebs with clear odorless serum. There is no emphysema.

Streptococcus or Staphylococcus Infection.

In these infections there is pus. There is no emphysema. The patient does not seem so ill. Blood culture in the latter infection may show the organism.

Gangrene Due to Obstruction, or Tearing of a Blood Vessel.

In this condition the part either becomes black, hard, dry, or moist, soft and ill-smelling. There is no emphysema, and the condition is limited strictly to the part supplied by the injured vessel. A history of injury is rarely obtained.

E. Infectious Diseases of Doubtful or Unknown Etiology

1. Vaccinia

(Vaccination, Cowpox)

Etiology.—This is the result of inoculating into the human being vaccine matter obtained from a human being or from cows—the latter being now universally used. The vaccine employed is believed to be the virus of smallpox which has become modified by passing through the cow.

Symptoms.—The symptoms are general and local. For three or four days after the operation the individual evinces listlessness, malaise and slight fever which may increase for two or three days, then disappear. Rarely is the individual very ill.

The local signs are the following: Immediately after vaccination, the site of operation entirely heals. On the second or third day, it becomes slightly reddened; a small papule forms, which soon becomes flattened and umbilicated, then rapidly forms a vesicle. Around the edge of this flattened umbilicated vesicle, redness of the skin occurs. One or more of the vesicles may coalesce, giving an appearance of a circular vesicle with an umbilicated, dark center surrounded by red, swollen, indurated skin (Fig. 36). The glands of the axilla may at this time become large and extended.

Development.—The height of the vaccination is about the seventh day, and after this if the part is not disturbed and it is not irritated, the vesicle gradually dries and becomes covered with a dark scale. During the height of the vaccination, the temperature varies, sometimes rising from 102° to 103° F. There is sometimes leukocytosis.

Vaccine Vesicles.—True vaccination is simulated by other conditions —among these the so-called spurious vaccinations. There may be a small, red tubercle which lasts for a considerable length of time and never becomes vesicular, or there may be one large, bullaelike vesicle with thin walls. Then, too, a true vaccine vesicle may become contaminated and form underneath it a deep and ulcerating sore on the arm, causing a large eroding ulcer, varying from the size of a dime to the size of a half dollar. When this occurs the site of vaccination is contaminated with some foreign organism. Occasionally there occur over the body during vaccination generalized vaccine vesicles. Severe gangrene may occur at the seat of the vaccination, which is also the result of contamination. Erysipelas may occur at the seat of vaccination and be extremely serious.

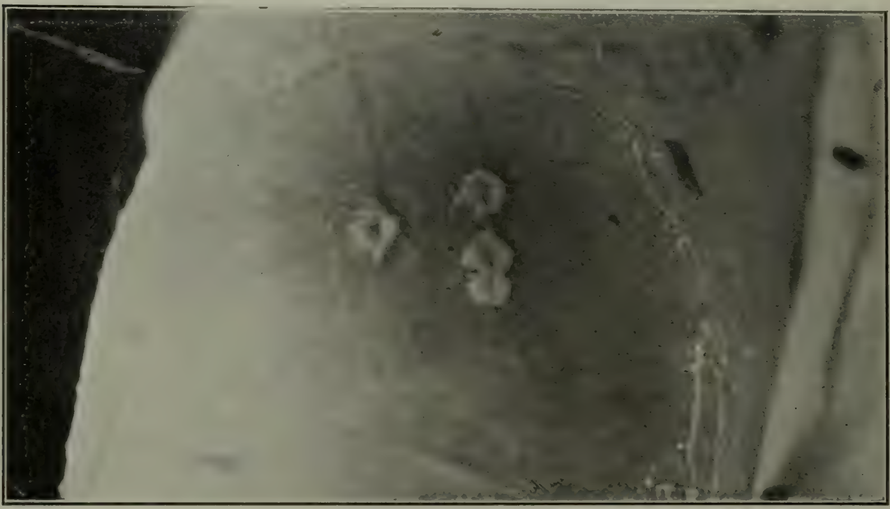

Fig. 36.—Successful Vaccination, 10th Day. (Kindness of Dr. Geo. Dock.)

Method of Vaccination.—One of the best methods for vaccination in the hands of the writer is as follows: The skin of the arm is carefully cleansed with alcohol; vaccine material is then placed upon the cleansed area and through the liquid a few scratch marks are made with a sterilized needle; the part is then rubbed either with the sterilized needle itself or with a sterilized bit of wood which often is supplied by the manufacturing company, and the part then covered with a bit of sterilized gauze and held in position with adhesive strips. This sterilized gauze is not disturbed for one week: it is then removed and a clean dressing put in its place.

Infection.—If these rules are followed, there will practically never be any contamination of the sore. The writer has seen fatal tetanus follow on two occasions; doubtless the infection was the result of dirt rubbed into the ulcerated area, because the tetanus occurred long after the vaccination. According to Dock, *hemorrhagic diathesis* may show itself first after a vaccination; *serious hemorrhage* sometimes occurs when leukemic individuals have been vaccinated.

2. Smallpox

(*Variola*)

General.—Smallpox is an acute infectious disease characterized by prodromal symptoms of chill, fever, headache, and backache, and on the third day by the appearance of the characteristic rash occurring about ten or twelve days after an individual *unprotected by vaccination* or a *previous attack* of smallpox has been exposed to the contagion of smallpox.

Cause.—The cause of smallpox is not yet definitely settled, though most authors believe the organisms to be bodies described by Guarniere, in 1892, and confirmed by Wasielewiski in 1897. Councilman, in 1904, in his article embracing the wonderful investigation made under his care, concludes that the bodies described are the real infecting organism of smallpox. He reaffirms his belief in his article in Modern Medicine, 1914.

Period of Incubation.—The time of incubation is usually ten to twelve days. This has been fairly definitely settled where cases have developed after a single exposure, lasting but a short time, to a case of smallpox.

Onset.—The patient is suddenly seized with a chill of greater or less severity. In children there is often a convulsion. Simultaneously with the chill a fever occurs which ranges from 102° to 103° F. At the same time the patient suffers intensely from severe headache and excruciating backache. These two symptoms distress the patient beyond any others. The throat is sore, there is nausea and vomiting; the patient feels extremely ill; there may be more or less delirium.

Eruptive Stage.—On the third day after the beginning of these symptoms a macular rash appears on the face, hands, wrists or legs, the seat of preference being the forehead near the hair line; they also occur early on the hands and wrists. Soon these macular spots change to papules, which can be likened to shot under the skin; they are hard and elastic. In a few hours the entire face, trunk and limbs are covered with a papular rash; if the rash is of unusual extent the papular vesicles coalesce. In two or three days the contents of the vesicles become purulent and are soon converted into pure pus. This change from papule to vesicle to pustule is rapid and affects the entire body, so that in a given time the entire rash is papular, or vesicular or pustular. One does not find a mixture of the varieties.

About the seventh day many but not all of the pustules become umbilicated—a depression on the top occurs. On about the ninth day the pustules rupture and the top of each individual pustule becomes covered with a crust.

Late Stage.—With the appearance of the eruption the temperature abruptly falls to normal. When the vesicles begin to become pustular the temperature again rises and remains high for about one week, when it falls by lysis. This is the so-called *suppurative fever.*

During this late stage of eruption, an odor due to decomposing pus pervades the patient's room and may be most nauseating. With the beginning of the eruption the skin of the face, and indeed of all portions affected, becomes much reddened, swollen and injected. The eyes are swollen and closed, the lips protrude, so that the individual becomes a loathsome sight and entirely unrecognizable.

In about two weeks the scales begin to separate, leaving behind a depressed reddened scar, the "pock mark"; the redness often lasts for days after the entire disappearance of the eruption. The later scar becomes white and remains throughout life.

Diagnosis.—This characteristic symptomatology is recognized by the merest tyro in medicine. Any physician who sees such a case can recognize it beyond question of doubt (Figs. 37, 38, 39, 40, 41, 42, 43).

It is in the *lighter cases,* called *varioloid,* however, that much difference of opinion may exist as to the true nature of the eruption.

This was particularly the fact in the epidemic which was so general in the United States during the past few years. Many thousands of cases occurred, particularly in the northwest, which gave rise to many diagnoses: CHICKENPOX, SYPHILIS, CUBAN ITCH—the latter not a pathological entity—and many other conditions.

However, in the lightest cases there is a more or less severe prodromal condition characterized by chilliness, headache and backache. On the third day or the beginning of the fourth a typical rash appears, first on the forehead near the hair line, then on the hands and wrists. There may be only a few pocks, but these are characteristic. They start as papules (according to Councilman, the eruption from the very first is a vesicle); these papules are hard, rolling under the finger like pieces of shot beneath the skin. They rapidly become vesicular to the ordinary observer, and on the third or fourth day, pustular. On the eighth or ninth day they rupture and soon are covered with a crust. The *pock is just as characteristic in the light cases as in the severe.* Umbilication may occur or may be absent in these light cases. The eruption is very apt to appear on the mucous membrane of the throat.

Symptoms.—*Backache.*—Backache is most severe. The pain is in the lumbar region and is usually complained of bitterly. The headache, backache, pain in the limbs, fever, chilly sensations in the absence of an epidemic of smallpox may readily be mistaken for the beginning of almost any febrile attack, especially LA GRIPPE or TONSILLITIS.

Vomiting.—Vomiting is common, sometimes severe and evidently depending upon the severity of the infection.

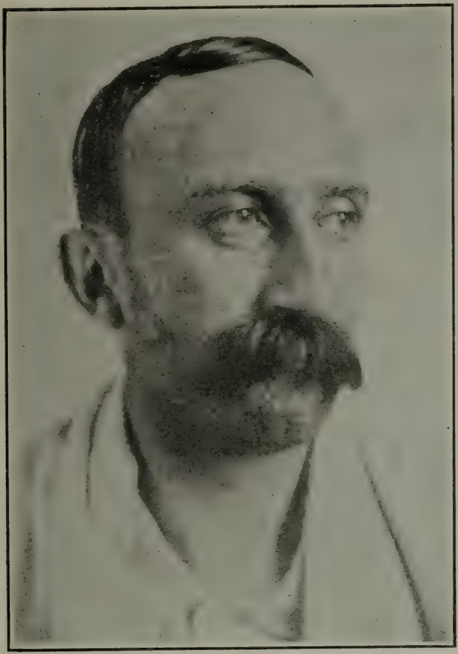

Fig. 37.—Smallpox; Third Day of Eruption. (Welch and Schamberg.)

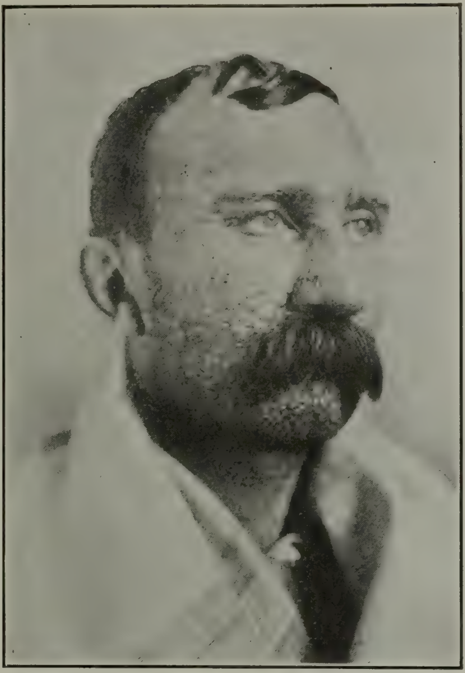

Fig. 38.—Smallpox; Fourth Day of Eruption. (Welch and Schamberg.)

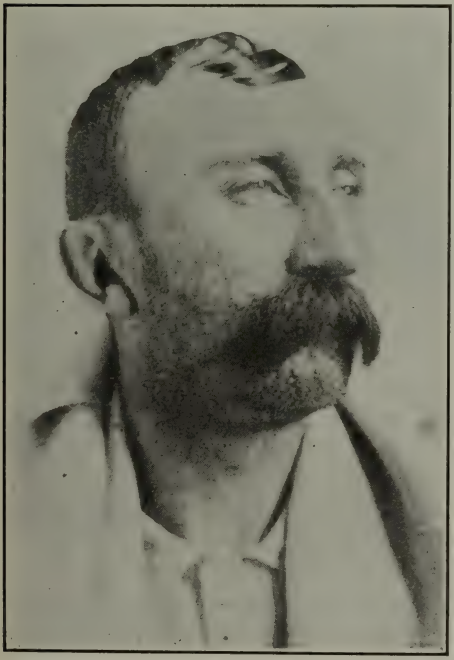

Fig. 39.—Smallpox; Sixth Day of Disease. (Welch and Schamberg.)

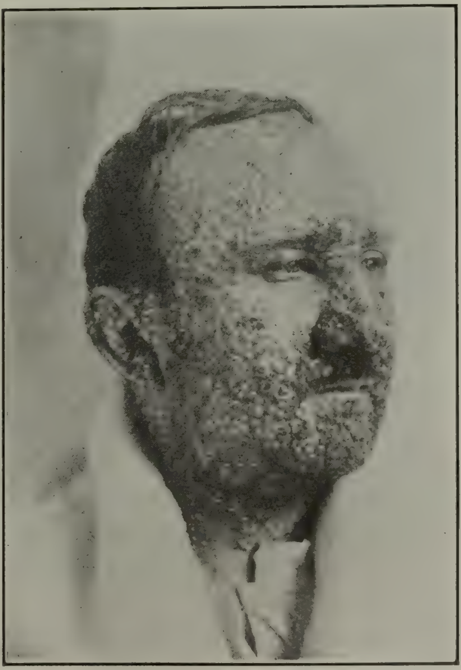

Fig. 40.—Smallpox; Eighth Day of Disease. (Welch and Schamberg.)

Prodromal Rashes.—These are of very common occurrence. Osler noted them in 13 per cent of his cases. They may resemble MEASLES or SCARLET FEVER, and be mistaken for them. A hemorrhagic rash occurs on the lower portion of the abdomen, on the thighs and axillae. This hemorrhagic rash may occur early in cases which afterward prove to be of a very mild type, though they are as a rule the forerunners of true hemorrhagic smallpox—one of the severest forms, almost universally fatal.

Temperature.—The temperature is quite characteristic. There is sudden rise of temperature usually coincident with the chill; this continues through the three days of prodromal symptoms, when, with the appearance of the rash, it rapidly falls to or near normal. After about twenty-four hours the temperature again rises and continues high for one or two weeks, when it falls by lysis. This latter is the so-called suppurative fever. It lasts as long as the pustular condition of the rash, and its severity corresponds with the extent of the rash.

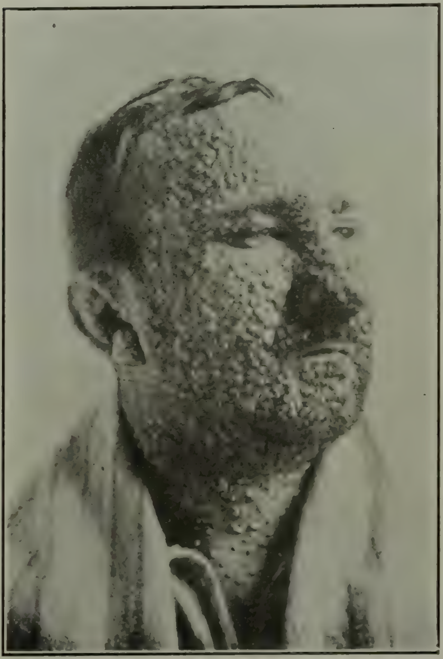

Fig. 41.—Smallpox; Tenth Day of Eruption. (Welch and Schamberg.)

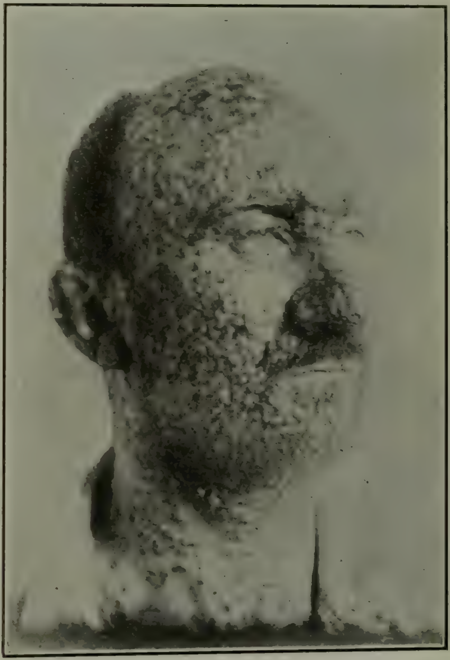

Fig. 42.—Smallpox; Sixteenth Day of Disease. (Welch and Schamberg.)

Rash.—The rash is often preceded by a diffuse erythema and occurs almost methodically on the third day after the initial chill. It is at first a reddish macule, rapidly becoming papular. This apparent papule is even in the beginning a vesicle (Councilman). It appears below the hair line of the forehead, on the hands and feet, choosing the portions of the body exposed to the air; the instep is frequently an early seat also, probably because the skin is more or less constantly irritated there. The rash appears over the whole body in a few hours. These papules have a "shotty" feeling; they roll under the finger like a small pea; they are elastic. On the second day of the eruption the papule becomes a distinct vesicle, the color of which is at first pinkish, and later pearly. The fluid is contained in small cavities in the epidermis. In one or two days a depression at the summit of the vesicle appears. This is the so-called umbilication, and is quite characteristic of smallpox, though in chicken-pox a circular depression occurs closely resembling this umbilication. About the sixth day of the eruption the vesicles become filled with pus, or rather the clear fluid of the vesicle becomes converted into pus. The degree, extent and characteristic features of the rash vary. In modified smallpox the spots may vary from one to a few hundred; they rarely if ever coalesce, and may never become umbilicated. This form is known as varioloid.

If the individual is not protected by vaccination or a previous attack of smallpox, the number of pocks is extensive: Schurling estimates 26,701 as the number in a moderately severe attack.

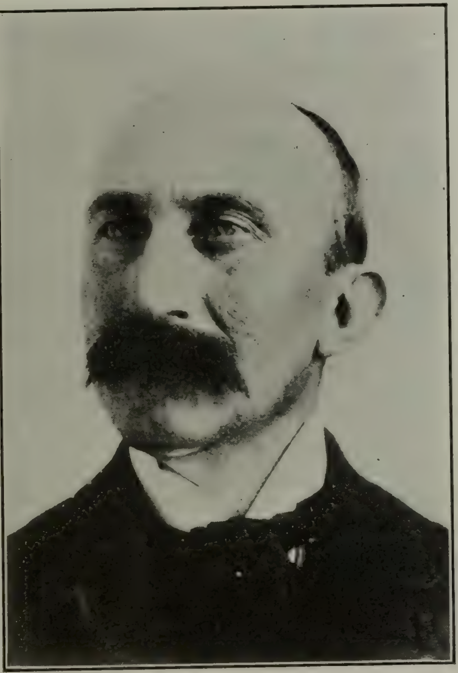

Fig. 43.—Appearance of Case After Recovery. (Welch and Schamberg).

The whole body may be covered and the pustules unite and become confluent. This is so-called *confluent smallpox*. If there is much hemorrhage under or between the vesicles or pustules the case becomes hemorrhagic, which is perhaps the most fatal form. In about two weeks the pustules rupture, the contents exude and the surface becomes coated with a thick crust. In neglected cases the pus collects under and between these crusts, gets on the bed clothes, and the whole body is redolent with a horrible odor.

Almost all cases of smallpox, whatever the severity, have complications—bronchopneumonia, nephritis, conjunctivitis are all common. The pock often occurs on the conjunctiva, but practically never upon the cornea. Toxemia is common.

Severe Cases.—In unvaccinated individuals occasionally the entire surface of the body is covered with a rash. This may be a marked hemorrhage into the vesicles, hemorrhagic smallpox, or the whole surface may be one continuous ulcer.

Mild Cases.—In certain cases of varioloid, which usually occurs in vaccinated individuals, and which may occur in individuals who have not been vaccinated, the initial symptoms may be either mild or severe; the rash consists of but few pocks which disappear without ulceration.

As late as 1912 an epidemic occurred in the coal regions of Pennsylvania which first confused the physicians under whose observation they came. Ninety-six out of the ninety-seven occurred in unvaccinated persons. Much harm is done by considering these mild cases to be some condition other than smallpox.

Conditions to be Differentiated from Smallpox

The diseases with which this condition is most frequently confounded are:

Scarlet fever
Measles
Chicken-pox
Cowpox
Syphilis
Drug eruption
Cerebrospinal meningitis
Impetigo contagiosum
Glanders
Pemphigus
Herpes
Acne.

In the very beginning, especially in isolated cases, smallpox may be mistaken for any of the febrile diseases which have an acute onset. In a few hours, however, or at least in a few days, a diagnosis can be made.

SCARLET FEVER.

In certain cases of malignant or fulminating scarlet fever there is a petechial rash which resembles the prodromal rash in hemorrhagic smallpox, but in the scarlet fever cases there is marked sore throat and swelling of the cervical glands which are not present in smallpox cases. This form of scarlet fever is usually rapidly fatal. The characteristic rash of scarlet fever is punctate, is red, and is not papular.

MEASLES.

Occasionally the rash in this disease has a papular form, but the Koplik's spots, coryza, conjunctivitis and bronchitis, which are an integral part of measles, never occur in smallpox. The early appearance of Koplik's spots on the buccal mucous membrane is a distinct and pathognomonic sign of measles, and the rash of measles lacks the shotlike feel of the papule of smallpox. The crescentric arrangement characteristic of the rash of measles must be looked for, while the appearance of the rash at the junction of the hair line and skin of the forehead is quite characteristic of smallpox. Grissolle's sign is sometimes of value. If the skin is grasped on either side of a papule and then made tense, the papule will disappear in most conditions except smallpox—in the latter disease it will remain above the surface of the skin.

CHICKEN-POX.

If the case is one of chicken-pox the prodromes will be slight or entirely wanting—though this is not universally the case. In a patient extremely ill with all the symptoms of chicken-pox, if the terrific headache and pathognomonic backache of smallpox are conspicuous by their absence, the diagnosis is decided by the character of the rash.

The rash of chicken-pox usually appears first on the trunk; the face, hands and throat may be affected later. It is at first a small red papule, but lacks entirely the "shot" feel of the papule of smallpox. It resembles a flea bite more closely than a hard papule. Within twenty-four hours this red spot becomes a prominent vesicle filled with a clear liquid. The wall of the vesicle is thin and easily broken; the base is often surrounded by a narrow red areola. Within an additional twenty-four hours the vesicles are converted into a spot covered with a scale; papules, vesicles and crusts cover various portions of the body at the same time, in contradistinction to smallpox, in which the rash is either papular, pustular or crusts over the various parts of the body at the same time.

SYPHILIS.

The pustular syphilitic rash is often mistaken for smallpox. The appearance of this symptom may be preceded by fever, malaise and general feeling of illness, but there is no accurate time relation between the prodromes and the rash as there is in smallpox.

In smallpox there are isolated pustules which rapidly soften, leaving a rounded superficial ulcer, while in syphilis the pustule does not break down so rapidly and leaves a deep excavated ulcer with ragged edges and sloughing base. The rash does not have the peculiar localization which smallpox does. In syphilis it may occur anywhere over the body and particularly on the trunk, while, as stated above, smallpox appears on the forehead, face, hands—the exposed portions of the body first. Syphilis of this character may occur late or early, so the coincidence of a local primary sore lesion does not always occur. The pustule of syphilis has a softened yellow summit. Syphilis gives a Wassermann reaction and spirochetes can be demonstrated. In syphilis there is an adenopathy, which is not present in smallpox.

Drug Rashes.

Drug rashes, especially those which are the result of copaiba, closely resemble the pustules of a mild case of smallpox with the following exceptions: there is no fever; the patient is ill only from the primary disease; there is no history of contagion; the patient may be fully protected by vaccination.

Cerebrospinal Meningitis.

The hemorrhagic form of smallpox or petechial form of prodromal rash might be mistaken for epidemic meningitis when petechiae are present in this disease, but a spinal puncture will settle the question. The spinal fluid of epidemic meningitis is cloudy and contains polymorphonuclear cells, some of which contain meningococci.

The rash of smallpox may become hemorrhagic. In some cases of smallpox the prodromal rash may be petechial, but the case lacks the stiff neck, Kernig's sign, and Babinski's sign of cerebrospinal meningitis.

Impetigo contagiosum.

Impetigo contagiosum occurs on the face and hands; there is but little systemic reaction. It occurs in vaccinated individuals. The vesicle is not preceded by a papule; it ruptures in twenty-four hours and is soon covered with a large crust.

Glanders.

Glanders occurs in vaccinated individuals. There is no papule preceding the pustule; the three stages of smallpox are not seen; there is the history of the individual having been in close communication with a diseased horse.

Pemphigus.

This is a serious disease characterized by large bullae, without the preceding papule, and without the resulting pustule. It occurs in vaccinated individuals.

HERPES.

Herpes is not preceded by a papule; the rash occurs along the branch of a diseased nerve; they are vesicles, in groups, and are not scattered over the entire body.

ACNE.

This is a chronic condition. Isolated acne pustules might be mistaken for a very mild case of smallpox, but the history of the regular appearance of a papule, pustular and scaling stage are wanting. The individual may have been vaccinated; this, of course, will influence the diagnosis.

3. Varicella

(Chicken-pox)

Definition.—Chicken-pox is an acute infectious disease most common in children. It is characterized by sudden onset, usually slight fever, and the appearance of a vesicular eruption appearing within twenty-four hours of the onset of the disease.

Onset.—The onset is sudden. Occasionally in weak individuals the disease is ushered in by a convulsion. In the usual case attention is frequently first attracted by the appearance of small vesicles, usually on the protected parts of the body. The fever may be high, though usually it averages from 100° to 102° F.; it quickly drops after the last crop of spots appears.

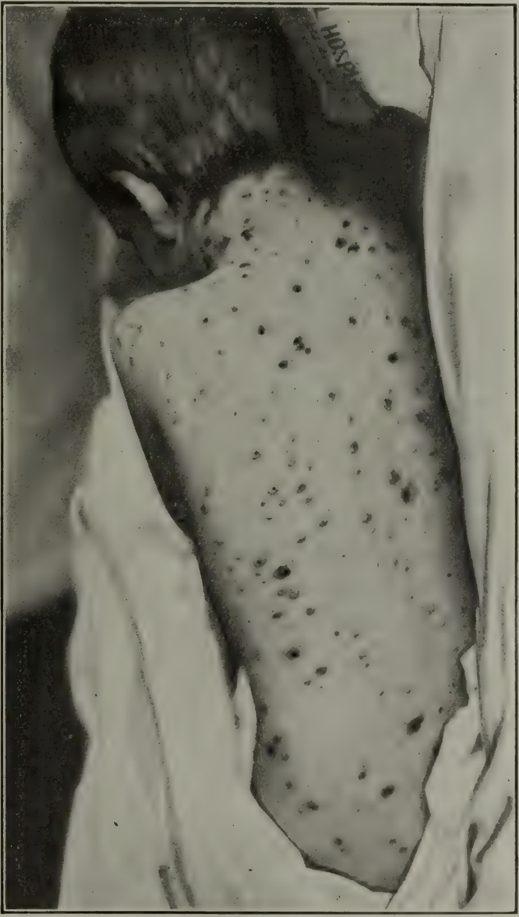

Fig. 44.—Rash of Chicken-pox. (Kindness of Dr. Jay Schamberg.)

Eruption. — The eruption first appears as a minute papule, becoming a vesicle in twenty-four hours; the vesicle, about two or three millimeters in diameter, is filled with perfectly clear liquid and the base is surrounded by a red areola. Within another twenty-four hours the fluid contents of the vesicles may have become slightly cloudy and the vesicles ruptured. The eruption usually appears first on the clothed parts of the body, though it may occur anywhere on the surface of the body (Fig. 44). It frequently occurs on the mucous membranes and particularly on the fauces. Attention is called to this by complaint of a sore throat. The rash has been observed on the conjunctiva, the larynx, in the urethra and vulva. The eruption may be seen in the macular, vesicular and crusting stage at the same time upon the same individual. Often there is inflammation and itching of the skin. If the patches become greatly irritated from itching or unclean clothing, they may suppurate and cause a secondary fever. MacCombie directs attention to a fact that on the forearms, legs, hands, arms and feet, the eruption is deep-seated and somewhat resembles smallpox, but examination of the rest of the body will show the typical small papule and vesicles.

Duration.—Within one week or ten days all signs of eruption may have disappeared.

Vaccination.—Vaccination against smallpox does not prevent varicella.

Conditions to be Differentiated from Chicken-pox

The condition may be mistaken for:

Smallpox
Herpes
Measles
Syphilis
Impetigo contagiosum
Dermatitis herpetiformis
Pemphigus
Molluscum contagiosum
Acne
Vesicular eczema.

SMALLPOX.

Varicella differs from smallpox first in the mildness of the symptoms, smallpox being as a rule a disease of severity, though in the vaccinated smallpox may give rise to few if any symptoms. Varicella occurs in vaccinated individuals as well as in unvaccinated persons; smallpox rarely occurs in the vaccinated.

The rash differs in every respect; that of varicella occurs on the first day, smallpox on the third day. Varicella rash rarely becomes pustular. Smallpox in the first two days of the eruptions is marked by a shotlike, papular swelling under the finger tips if palpated. The rash of varicella does not, except in very rare cases, become umbilicated or markedly prominent; it does not pit the skin. A certain form of varicella, called varicella gangrenosa, may cause some confusion, but here there is not the shotlike hardness of the rash, as there is in smallpox.

Herpes.

Varicella may resemble herpes somewhat, but the latter has certain exceptional symptoms. The vesicles of herpes are grouped along the line of the nerve the ganglion of which is affected. There may be constitutional symptoms. Pain is a symptom of herpes, which is not present in varicella.

Measles.

The constitutional symptoms of measles—fever, cough, coryza—distinguish it from varicella. The rash of measles is always a very flat macular papule. There are no Koplik's spots in varicella; they are present in measles.

Syphilis.

In the papular and pustular stage of syphilis there may be some difficulty in diagnosis, but there is always the initial sore; spirochetes can be demonstrated; the Wassermann reaction is apt to be present. There is not the clear vesicle preceded by the small papule characteristic of varicella.

Impetigo contagiosa.

This disease occurs mostly on the hands and face. It begins with a vesicle which enlarges rapidly, breaks down and is covered with a thick scale. These lesions lack the "dew drop" character of chicken-pox.

Dermatitis herpetiformis.

According to Duhring, the lesions in this disease are likely to form in groups. The course of the case is chronic, with sudden exacerbations. The rash differs at different stages of the disease. The vesicles are arranged in groups—unlike the rash of chicken-pox.

Pemphigus.

Acute pemphigus, according to Fox (Allbutt's "System of Medicine," volume IX, page 429), occurs with a febrile reaction—the temperature being high. The bullae are large and rise from the skin without any erythema. In true pemphigus the bullae vary in size from that of a split pea to an orange. It is a grave disease which frequently ends fatally—differing entirely from the slight ailment, chicken-pox. Chicken-pox might be mistaken for it when it occurs in the gangrenous type.

MOLLUSCUM CONTAGIOSUM.

Molluscum contagiosum differs from chicken-pox in that the tumors are not transparent. While it is contagious, it is chronic in its course. The lesion is a true tumor.

ACNE.

Acne differs from chicken-pox in the facts that the disease is chronic, is not contagious, and without exception the lesions become pustular and are often surrounded by an indurated area.

4. Scarlet Fever

General.—Scarlet fever is an acute infection characterized by sudden vomiting, high fever, sore throat and a scarlet rash occurring within twenty-four hours, and desquamation of the skin after several days or weeks.

Organism.—The actual source of contagion is unknown. Hektoen, in extensive observations, concludes that the Streptococcus pyogenes is certainly not the organism involved. The protozoanlike bodies of Mallory have been found by other observers but never in skin removed during the life of the patient. The disease is spread by means of the nasal and throat discharges, and perhaps by desquamation.

The Attack.—Attacks vary markedly in type, not only in individuals during an epidemic, but epidemics differ in the virulence of cases.

General Symptoms.—The onset as a rule is sudden. The first symptoms may be a convulsion, vomiting, high fever, with death in twenty-four hours, or the first and only symptom noticed may be a red rash. Cases vary between these two extremes; there are all degrees of severity.

The symptoms vary during the first twenty-four hours of the attack, depending upon the *virulence of the form of the disease* and the *resistance of the individual attacked.*

ATYPICAL SCARLET FEVER.—*Fulminating Form.*—In the fulminating cases the patients may die before a diagnosis can be made, unless, indeed, the attack occurs during an epidemic. In these cases the patient is instantly seized with a convulsion, high fever reaching 104° to 106° F., unconsciousness, rapid running pulse and rapid respirations. The back of the throat is usually injected and sprinkled with a dark punctate rash. Over the body, before death, there is usually a very sparse rash, which may be petechial, but at the same time retains the punctate appearance of the normal rash. The patient dies in collapse within a few hours.

Malignant Form of Scarlet Fever.—Then there is a form extremely severe, in which the disease is ushered in by high fever, convulsions, vomiting and severe sore throat. The patient is extremely ill from the beginning; the temperature ranges from 104° to 105° F., the rash is profuse and bright red, the throat sore and covered with a more or less extensive membrane; the tongue is dry, the pulse and respiration rapid. If the patient survives, desquamation begins within five or six days and the entire body may be covered by scales in a few days, though the desquamation is not complete until three, four weeks—perhaps more—have elapsed. The bowels may be extremely loose and a fetid diarrhea occurs.

Anginose Form.—There is a form in which there is a severe angina without any eruption. This can be diagnosed only if it occurs during an epidemic.

Typical Scarlet Fever.—In the average case of scarlet fever, the patient is suddenly ill, vomiting occurs, and there is very sore throat. Usually within twenty-four hours the rash makes its appearance, first on the chest, or in the axillae and groins. It rapidly spreads over the entire body. It becomes covered with minute red spots. The skin between these spots is the seat of a scarlet erythema. In severe cases the whole skin is fiery red with deeper red punctate spots appearing all over the body. Sometimes a miliary rash occurs over the body superimposed on the red rash. The face is flushed; the mouth surrounded by an area of anemia; the tongue is heavily coated with white fur, the prominent red papillae showing through the coating and giving the appearance, as Flint long since described, of the entire tongue being sprinkled with Cayenne pepper. Within a few days the entire epithelium of the tongue desquamates and the whole tongue covered with prominent and swollen papillae becomes bright red—the "strawberry tongue." The glands at the angle of the jaw are involved early, often forming huge masses under the ear.

In a few days to a week desquamation begins. It is seen early about the skin at the edges of the nails. This desquamation occurs over the whole body; on the chest it may be in small, even, branny scales, on the palms of the hands and the soles of the feet the entire epidermis may be lifted, causing large flakes, and in some instances a cast of the entire hand or foot.

The *urine* contains albumin, and perhaps casts in many cases. Each symptom here described may be absent and the case may be a mere febrile attack, lasting but a few days, with scarcely any discernible rash; or the rash may be the first symptom of the attack to attract the attention of the parents. In these instances it is of the greatest importance to make a proper diagnosis, both from the standpoint of the individual affected and from that of public health. When there is doubt the case should be diagnosed scarlet fever, until proven to be some other disease.

Special Symptoms.—*Fever.*—The temperature rises rapidly, frequently it is at its height when the patient is first seen. The temperature ranges from 104° to 106° F. in fulminating and severe cases. In moderate cases the temperature reaches its height of 103° F. or 104° F. within forty-eight hours, remains high for five to nine days, and then rapidly within forty-eight hours reaches normal, to remain so unless some complication occurs.

The Eruption.—The scarlet rash from which the disease obtains its name occurs in the vast majority of cases within from six to twenty-four hours after the beginning of the active fever. It covers the entire trunk and limbs, and is absent or very sparse on the face; it is more brilliant in the axillae and groins. It consists of small punctate spots, each point being surrounded by an area of erythema of bright hue; usually the erythematous areas coalesce, the entire body is red, but the deeper red punctate spots can always be distinguished in the midst of the general redness. In addition to this red rash which is usually observed there are vesicles at the site of the hair follicles scattered over the body more or less profusely. According to Schamberg, these vesicles are present in the vast majority of cases of scarlet fever and are more copious in severe cases. In the colored race the eruption on the skin gives a bluish appearance, which can scarcely be described, to the entire body.

The face in scarlet fever is flushed, usually free from rash, with a white anemic ring surrounding the mouth. This white ring about the lips and mouth is rather characteristic of scarlet fever.

Sore Throat.—Usually the soreness of the throat is one of the first symptoms of which complaint is made. On examination, the fauces, tonsils and entire pharynx are found to be bright red. Soon there appears over the bright red surface plaques of white, rather "stringy" in appearance. If a secondary infection occur, the exudate may form a membrane not to be differentiated from diphtheria, save by culture. Occasionally a true diphtheritic membrane occurs.

Desquamation.—Peeling of the skin is present in scarlet fever, almost without exception. It frequently begins on the hands, feet and root of the nails; it persists for weeks. On the body it may be in small branny scales, or as is much more usual, in large patches, irregular in outline. On the hands and feet the scales are almost always large, and can be peeled off in great patches, occasionally forming true casts of the hands and feet.

Adenitis.—Inflammation and swelling of the glands at the angle of the jaw is the rule; usually this swelling subsides with the disappearance of the fever, but the glands may suppurate and form one of the most severe forms of complication.

Complications

The most frequent complications are suppuration of the middle ear, nephritis, suppuration of the cervical lymphatics, gangrenous sore throat, pneumonia, endocarditis and pericarditis; arthritis occurs but less frequently than the other complications.

Otitis Media.—Otitis media occurs, according to McCollom, in 18 per cent of 5,000 cases observed in the South Department of Boston City Hospital; it may be a serious threat to the life of the patient, or it may eventuate in little more than a purulent discharge which soon disappears. When the condition occurs early in the course of the disease, often little disturbance is caused. Deafness is doubtless always present, as an initial sign; pain may be entirely absent. Frequently the first evidence of otitis media is a purulent discharge from the ears. For these reasons the ears of every scarlet fever patient must be carefully watched and proper precautions taken. From the inflammation of the middle ear, acute inflammation of the mastoid cells may occur, giving rise to a true mastoiditis and a consequent brain abscess. Tenderness over the mastoid demands attention at once—if the attendant is not skilled in surgery of the ear, one so skilled should be called at once. Mastoiditis usually occurs late, but the author has notes of one case in which the mastoid was affected on the third day.

Nephritis.—Nephritis is a common accompaniment of the disease. Scarcely a case of any severity escapes without having albumin and perhaps a few tube casts found in the urine. These elements, however, are simply due to the mild nephritis, and may be found in almost any severe febrile attack.

A true inflammation of the kidneys occurs frequently, however. It may occur as a complication of a severe attack, being characterized by smoky, scant urine or almost complete anuria. The urine contains much albumin and many tube casts, dark granular and blood casts, also free blood. Nephritis may also occur as an actual sequel or very late in the course of slight or moderate attacks. Death may occur from uremia. Frequently the first indication of an attack is a uremic convulsion, in a child with general dropsy—scarlet fever may never have been recognized.

Suppuration of the Cervical Glands.—This may occur sometimes in the course of the disease, sometimes later. This condition is a fertile cause of mortality.

Gangrenous Sore Throat.—Gangrenous sore throat is a serious complication due to a secondary infection; it may occur in both slight and severe attacks of fever. The writer has notes of more than one case in which perforation of the soft palate was about the only serious symptom in an otherwise mild case.

Endocarditis—Pericarditis.—Endocarditis and pericarditis occasionally occur. They are recognized only by methodical examination of the heart. A case which does not show the normal fall of the temperature or where the fever has fallen to normal and then risen is very apt to be due to one of these various complications—hence the necessity of a careful daily examination of the lightest case, including several examinations of the urine.

Scarlatinal Arthritis.—Scarlatinal arthritis is a more or less generalized arthritis, not a true rheumatism, but an expression of the toxemia.

Conditions to be Differentiated from Scarlet Fever

The condition must be diagnosed from:

MEASLES
RUBELLA
DRUG RASHES
ERYTHEMA INFECTIOSIS.

For special symptoms, see below, under *Measles*.

5. Measles

(*Morbilli*)

Organism.—The organism which is the cause of this disease is unknown.

General Symptoms.—Measles is an acute infectious disease characterized by catarrhal inflammation of the nasal and bronchial mucous membrane, Koplik's spots occurring on the mucous membrane of the cheeks, and a characteristic skin eruption appearing on the third day of the attack. The symptoms are drowsiness, fever, coryza, conjunctivitis and bronchitis accompanied by an annoying dry cough.

Course of the Disease.—According to Rurah, there is a *leukocytosis* in the beginning, which rapidly falls to normal.

Koplik's Spots.—If the *mucous membrane* of the cheeks be examined on the first or second day, there will be discovered just opposite the lower molar on both cheeks, minute white spots on bluish, slightly elevated areas surrounded by a red areola. When discovered these are pathognomonic of measles. They occur at least forty-eight hours before the characteristic rash of measles on the skin. In the author's experience they are difficult to recognize under certain conditions, and must not be confounded with the aphthous ulcer and spots due to oidium albicans. Aphthous ulcer is a true ulceration which is depressed and painful, of a size varying from a pin's head to one-third of an inch in diameter.

Rash.—On the third day the *fever falls to near normal*, coincident with the appearance of the rash, manifested first in the throat and then on the face and neck. It looks not unlike flea bites. These spots rapidly multiply and become grouped in concentric masses. Within twenty-four hours the whole surface of the skin is covered with a rash, but the large individual spots can be distinguished and usually the concentric grouping is easily recognized. The throat is the seat of redness on the first or second day. Early in the third typical red spots appear on the uvula and soft palate.

Within a few hours of the appearance over the face, the rest of the skin of the body becomes affected—almost the whole body appears red; however, there are always areas of healthy skin between the parts affected. Following the appearance of the rash, the fever recurs, remaining for three or four days, during which time the patient may be extremely ill. During this process the cough and coryza continue, while gradually the fever diminishes.

The mucous membrane of the bowels may be affected and the patient suffer with *diarrhea* or *dysentery*. In some cases the *exhaustion* is extreme

The fever and catarrhal symptoms gradually subside, the rash disappears, and at the end of a week or ten days the patient is quite well though the catarrhal symptoms often remain much longer.

The *desquamation* is usually universal, but slight. The scales are branny, and difficult to distinguish; *they are unlike the large scales of scarlet fever.*

Virulent Cases.—Certain cases of measles are extremely virulent in type—this is particularly the case when men on shipboard or soldiers in barracks are affected, or when a community previously unaffected is attacked. Here the lowered vitality of those attacked, or the entire lack of immunity, causes the disease to be much more severe. The blood shows no change except when some suppuration occurs such as in the middle ear or pneumonia.

Complications—Symptoms.—LUNGS.—The lungs are constantly the seat of a *bronchitis* of more or less severe type; this gives rise to constant distressing cough, often unproductive. Occasionally there is a profuse bronchorrhea which in very amount of the discharge is a threat both to the comfort and safety of the patient. Irregular fever, leukocytosis, continued cough with physical signs of dullness in the lungs and blowing breathing, or a spot over which fine râles are persistently heard, are signs of a *pneumonia.*

LARYNX.—*Laryngitis* is another complication of importance, usually characterized by a strident cough with a hoarse voice. Occasionally there is some difficulty in breathing, but this is rare unless the larynx becomes the seat of a membranous exudate. This latter may be a diphtheritic exudate, or a true membrane without the presence of diphtheria bacilli.

EYE.—The *conjunctivitis* which is present almost without exception, causes more or less severe *photophobia.* This may be extreme, the patient being unable to allow the least amount of light in the room. The presence of extreme photophobia is a fair indication of the presence of a *corneal ulcer.* Careful examination of the eyes should be made frequently in order that a severe keratitis may be forestalled.

EAR.—Inflammation of the middle ear is frequent. The constant inflammatory condition of the nasopharynx gives rise to the extension into the middle ear; suppuration often occurs, but the *middle ear disease of measles does not give rise to the bone complications which are so common in scarlet fever.*

INTESTINES—COLON.—*Enteritis* and *colitis* are common with all their usual symptoms; in young children these may be of grave import.

Diseases to be Differentiated from Measles

RUBELLA.

Rubella has scarcely any fever; the individual is not ill; the posterior cervical glands are enlarged. The rash is usually in distinct spots, but it may exactly resemble that of scarlet fever or measles. The throat is not sore. Desquamation occurs but it is difficult to observe.

SCARLET FEVER.

Scarlet fever begins suddenly. The rash appears in the first twenty-four hours. There is no conjunctivitis, coryza or bronchitis before the rash; there are no Koplik spots.

The throat of scarlet fever is brick red; often there is grayish exudate; the cervical glands are enlarged. There is tendency to nephritis.

The rash starts on the chest in the axillae and groins; it is punctate, the whole body becoming red and covered with a flush between the spots of rash. It is not concentric.

The tongue is first covered with a thick white fur, with prominent red papillae showing through it. In four or five days the coat disappears; the papillae, however, remain prominent (strawberry tongue).

Desquamation is evident, the skin often separating in large flakes, and often casts of the fingers and toes occur.

The temperature is high and often becomes septic in type. There is leukocytosis.

SMALLPOX.

Smallpox may easily be mistaken in the early stages, notwithstanding the great difference. Often the primary erythema and fall of temperature at the beginning cause the error. In smallpox there is often an initial chill; there are no catarrhal symptoms; there is more severe headache and backache. There are no Koplik's spots; the preliminary rash of smallpox is larger. The characteristic rash is papular.

Typhus Fever.

In typhus fever the general symptoms are more intense; the rash is petechial; there are no catarrhal symptoms. There is the history of infection by lice.

Influenza.

Influenza may have the catarrhal symptoms of measles, but there is the absence of Koplik's spots, besides the characteristic rash does not appear.

Drug Rashes.

Drug rashes may resemble measles, but the absence of catarrhal symptoms, and Koplik's spots and the fact that the rash rarely appears in the throat, distinguish it from measles. Quinin or antipyrin rash may exactly resemble measles, except for the history of drug-taking and absence of the above symptoms.

Syphilis.

Syphilis is characterized by the initial sore, the gland involvement; there is absence of catarrhal symptoms and of Koplik's spots. The Wassermann reaction is present.

6. Rubella

(*Rötheln—German Measles*)

Characteristic Features.—This disease is characterized by a rash, red in color, which suddenly appears upon the face, trunk, limbs and mouth, without the accompaniment of any grave general symptoms.

Forchheimer describes the mouth rash as follows: "It consisted of a macular, distinctly rose-red eruption upon the palate and the uvula, extending to but not upon the hard palate. The spots are arranged irregularly, not in groups, of the size of large pin heads, very little elevated above the level of the mucous membrane, and do not seem to produce any reaction upon it."

Sometimes the rash on the body resembles measles, sometimes scarlet fever. Sometimes there is severe fever, and often whole epidemics occur which include but few cases with symptoms of any moment except the rash. Edwards, however, reported fatal cases.

Usually the glands over the body are involved, and there are chains of these enlarged glands running up the back of the neck. There is very little sore throat—no exudate upon it. The rash is uniform over the whole body; the individual spots are somewhat larger than those of scarlet fever and the erythema of the skin is less intense. There is a very fine, almost indiscernible desquamation.

Complications.—There are few if any complications.

Conditions to be Distinguished from Rubella

The chief interest in the disease lies in confounding it with:

Scarlet fever
Measles.

SCARLET FEVER.

Scarlet fever is accompanied by high fever, sore throat, enlargement of the cervical glands which may suppurate; often nephritis occurs. The rash appears on the chest first; it is punctate, but there is a general erythema covering the whole skin. It is in light cases of scarlet fever, however, that the greatest difficulty is encountered. In these cases the child may be scarcely ill but there is usually sore throat; the rash is punctate and very red; there is much desquamation and the characteristic tongue. Diagnosis may have to be delayed until the tongue shows characteristic signs or there is desquamation.

MEASLES.

In measles there is always the signs of coryza, bronchitis and conjunctivitis which are wanting in rubella. Koplik spots are present.

Summary of Differentiation

Scarlet fever, measles and rubella must be differentiated from each other and from the various other erythemata and from diphtheria.

Initial Symptoms.—*Scarlet fever* is ushered in by vomiting, fever and sore throat. The rash appears within twenty-four hours after the beginning of the onset.

Measles begins with all the symptoms of an acute coryza and bronchitis, plus fever, the rash appearing on the third or fourth day.

Rubella begins with few or no symptoms, the first sign frequently being the rash and swelling of the glands.

Rash.—The rash of *scarlet fever* occurs within twenty-four hours of the beginning of the disease. The rash is punctate and gives a general scarlet blush to the skin. Connecting these punctate points is a scarlet erythema. This usually appears first on the trunk and is always accentuated in the axilla, groins and in the flexure of the limbs.

The rash of *measles* appears first upon the face and neck, as distinct red spots, rapidly forming into crescentric groups. The rash may cover the entire body, but the distinct spots are slightly raised, or at least accentuated in color, giving the skin a rough appearance.

The rash of *rubella* is notoriously polymorphous, sometimes punctate, resembling chiefly that of scarlet fever, at other times large and distinct like measles. It appears as small red areas on the uvula and soft palate.

Throat.—*Scarlet fever* is marked by a distinct sore throat; the mucous membrane is scarlet in color. Later there may be an exudate, usually in shreds over the tonsils, sometimes resembling that of diphtheria.

The throat of *measles* is distinctive in the first twenty-four or forty-eight hours. Koplik's spots—red raised patches with bluish summit, capped with a white apex—are seen on the buccal mucous membrane. These spots are pathognomonic. On the second or third day there appears on the uvula and soft palate, red raised spots which are a part of the general rash appearing on the skin. *Rubella* has no sore throat.

Tongue.—The tongue of *scarlet fever* is at first coated white, red papillae showing plainly through the white fur, as though Cayenne pepper were spread over it. In from four to six days the epithelium desquamates, leaving the tongue bright red, with the papillae prominent—the strawberry tongue.

The tongue of *measles, also rubella,* may be slightly or heavily coated, but there are distinctive features.

Fever.—The temperature of *scarlet fever* is high in the beginning and may remain so from four days to a week or eight days, when it gradually falls to normal.

The temperature of *measles* is high for the first twenty-four hours; it then falls to nearly normal for twenty-four hours; it then rises again and reaches its acme with the appearance of the eruption; it then either remains high for two or three days or rapidly falls to normal.

The fever of *rubella* is very slight, or the temperature may be normal.

Blood.—The blood of *scarlet fever* shows a leukocytosis which persists after the disappearance of the fever. The *blood of the other conditions* may have a leukocytosis in the beginning, but it disappears unless some secondary complication arises.

Erythema.—*After the use of antitoxin* the erythema which occurs is not unlike the erythema of measles in certain instances. There are no constitutional symptoms. The knowledge that serum has been administered will help in the diagnosis.

In erythema contagiosum the only symptom is an erythematous eruption not unlike that of scarlet fever; the desquamation which occurs is large and flaky; the tongue is unchanged.

One may see, occasionally, a red erythematous blush over the skin of children who are hot and flushed for any cause—occasionally this happens when they have been covered with a cheap blanket or quilt of red color, which color may be imprinted on the skin—but in this instance the disappearance of the supposed rash can easily be accomplished by washing.

7. Infective Parotitis

(Mumps)

Microörganism.—The microörganism is unknown.

Characteristic Features.—Epidemic parotitis is an infectious disease. Its chief characteristic is a sudden painful swelling of one or both parotid glands, usually accompanied by fever and slight general symptoms.

Onset.—The disease begins suddenly. Frequently the first symptom is stiffness of the jaws rapidly followed by swelling of one or both the parotid glands.

Course of the Disease.—If the condition begins with the swelling of one gland only, usually in the course of from twenty-four hours to five days, the second gland becomes swollen. Occasionally the other salivary glands, the sublingual and the submaxillary, are involved and cases are on record where the lacrimal gland was involved. The disease is usually confined absolutely to the region of the parotid gland. Frequently the swelling begins immediately below the lobe of the ear, extending forward and around it, so that the latter is pushed forward. The gland is hard, tense and easily palpable, is not reddened, and there is no fluctuation. Opening and closing the mouth often give considerable pain. As has been said before, there is usually fever. Sometimes the condition is severe, begins with a chill, and is followed by a high fever which reaches 102° or even 104° F., lasting three or four days and disappearing suddenly. The whole neck may be swollen.

Late Stage.—In the course of the disease or the following days, after convalescence, there sometimes is an *orchitis*. With the onset of the orchitis there is usually increase of fever and what appears to be grave septic condition, and the patient may develop a typhoid state. This is unusual—the most serious occurrence as a rule is a painful testicle on the side affected. In girls there may be vulvovaginitis and swelling of the breasts—no case of involvement of the ovaries is on record. The pancreas has been involved.

Diseases to be Differentiated from Infective Parotitis

This condition may be differentiated from the following:

Secondary parotitis
Swollen lymphatic glands
Trichiniasis
Parotitis following dysentery
Mikulicz's disease
Typhoid fever
Puerperal sepsis and operations.

SECONDARY PAROTITIS.

The occurrence of a parotitis in any well-developed disease, such as typhoid fever and pneumonia, or after an operation, is proof, as a rule, that the parotitis is secondary to the disease or the operation. One might be in a quandary after an operation or after one of the diseases mentioned, where the patient has been exposed to a case of mumps—but this is extremely rare. There is no difference in these cases of secondary parotitis in the appearance of the gland from that in the real disease mumps. Occasionally in secondary parotitis there is suppuration in or about the gland.

SWOLLEN LYMPHATIC GLANDS.

Swelling of the lymphatic glands at the angle of the jaw, ought never to be mistaken for parotitis, because by palpation of the swollen area, one can definitely outline either the lymphatic glands or the parotid gland. Occasionally there may be a little difficulty caused by the fact that a lymphatic gland immediately overlying the parotid may be swollen.

TRICHINIASIS.

In trichiniasis the masseter muscle sometimes is so affected by the implantation of the trichinae that it might be mistaken for the parotid gland.

MIKULICZ'S DISEASE.

This is an enlargement of the salivary glands and lacrimal glands, chronic in course. When the parotids are enlarged the condition may resemble ordinary mumps with this exception—there is no fever. The case is likely to have the lacrimal glands enlarged and Mikulicz's disease is chronic in its course.

8. Typhus Fever

General Statements.—This is an infectious disease, heretofore considered highly contagious. It has been one of the world's scourges, but now is extremely rare except in certain parts of the country. The typhus fever of Mexico is still prevalent and is the same disease as the typhus fever of early times and of continental Europe. Mild cases are often present today in the United States; they were called to the attention of the profession by Brill.

Mild Form.—The set of symptoms described by Brill are called *Brill's disease,* but observations and experiments by Anderson and others have proven beyond peradventure that *Brill's disease is really a mild form of typhus fever.* The name should not be used and all such cases should be given the proper cognomen—typhus fever.

Origin.—Typhus fever occurs in epidemics, and is carried from one individual to another by the body louse and probably also by the head louse as proven by Anderson, Goldberger and Nicolli.

Incubation.—During the incubation there are no characteristic symptoms, but malaise, slight indisposition and other symptoms common to the beginning of almost all infectious diseases are seen.

Onset.—The actual onset of the disease is sudden, with chill, headache, and high fever as the main symptoms.

Even in the beginning the face is flushed and dull red; the patient lethargic and evidently the victim of a severe infection. The temperature rises very rapidly and reaches 104° to 105° F. within the first day.

Course of the Disease.—The temperature continues high during the course of the disease, lasts about one week or ten days and ends abruptly, the defervescence never lasting more than forty-eight hours.

The patient becomes prostrated. Delirium of a mild degree follows and finally becomes more violent. On the third or fourth day a petechial rash appears first on the abdomen and then over the whole body. If the case is severe the rash often increases and becomes more hemorrhagic, the patient becoming quite or almost comatose.

Eruption.—The eruption and course of the disease is thus described by Osler:

> "From the third to the fifth day the eruption appears—first upon the abdomen and upper part of the chest, and then upon the extremities and face; occurring so rapidly that in two or three days it is all out. There are two elements in the eruption: subcuticular mottling, 'a fine irregular, dusky red mottling, as if below the surface of the skin some little distance, and seen through a semi-opaque medium' (Buchanan); and distinct papular rose spots which change to petechiae. In some instances the petechial rash comes out with the rose spots. Collie describes the rash as consisting of three parts: rose-colored spots which disappear on pressure, dark red spots which are modified by pressure, and petechiae upon which pressure produces no effect. In children the rash at first may present a striking resemblance to that of measles and gives as a whole a curiously mottled appearance to the skin. The term mulberry rash is sometimes applied to it. In mild cases the eruption is slight, but even then is largely petechial in character. As the rash is hemorrhagic, it does not disappear after death. Usually the skin is dry, so that sudaminal vesicles are not common. It is stated by some authors that a

distinctive odor is present. During the second week the general symptoms are much aggravated. The prostration becomes more marked, the dilirium more intense, and the fever rises. The patient lies on his back with a dull, expressionless face, flushed cheeks, injected conjunctiva, and contracted pupils. The pulse increases in frequency and is more feeble; the face is dusky, and the condition becomes more serious. Retention of urine is common. Coma-vigil is frequent, a condition in which the patient lies with open eyes, but quite unconscious; with it there may be subsultus tendinum and picking at the bed clothes. The tongue is dry, brown, and cracked, and there are sordes on the teeth. Respiration is accelerated, the heart's action becomes more and more enfeebled, and death takes place from exhaustion. In favorable cases about the end of the second week occurs the crisis, in which often after a deep sleep, the patient awakes feeling much better and with a clear mind. The temperature falls, and although the prostration may be extreme, convalescence is rapid and relapse very rare. This abrupt termination by crisis is in striking contrast to the mode of termination in typhoid fever."

Conditions to be Differentiated from Typhus Fever

Typhus fever may be mistaken for the following diseases:

Typhoid fever
Smallpox
Scarlet fever
Measles
Relapsing fever
Purpura
Septicemia
Rocky Mountain spotted fever
Cerebrospinal fever
Malarial fever.

TYPHOID FEVER.

Typhus fever is easily distinguished from typhoid fever by its abrupt onset, its shorter course, its recovery by crisis, the character of its rash. The presence of diarrhea, of enlarged spleen, the gradual fall of temperature, the Widal reaction, a positive blood culture for typhoid bacilli, and a leukopenia occur in typhoid fever.

SMALLPOX, SCARLET FEVER AND MEASLES.

There may be difficulty at the beginning in distinguishing typhus from smallpox, scarlet fever and measles—they all have the abrupt beginning of a febrile condition. The measlelike eruption sometimes found at the beginning of typhus fever makes the diagnosis difficult. However, in a case of smallpox, after three days the rash appears, papular in type and rolls like shot under the fingers, while the rash of typhus is petechial.

The greatest difficulty is encountered when the rash of smallpox is hemorrhagic. There is also a sudden drop of temperature in smallpox at the appearance of the rash, which does not occur in typhus fever. In scarlet fever the rash appears in twenty-four hours and is of the characteristic type of punctate spots with erythema. There is sore throat. In measles the Koplik's spots and the accompanying bronchitis, not present in typhus, and the situation of the rash on the face, make the diagnosis.

RELAPSING FEVER.

Relapsing fever may be easily distinguished by examination of the blood, in which spirochetes may be found.

PURPURA.

Purpura might possibly be mistaken for typhus fever were it not for the fact that there is no characteristic febrile condition in typhus fever. Insect bites might possibly be mistaken in the course of some other fever, but these may be easily diagnosed, as McCrae suggests, by the presence of the scar from the bite in the center of the lesion.

SEPTICEMIA.

Certain forms of septicemia with abrupt onset and petechial rash might be mistaken for typhus, but there are usually the evident causes for sepsis, such as a wound, uterine disease or heart disease, which of course are not present in typhus fever.

ROCKY MOUNTAIN SPOTTED FEVER.

This disease occurs in certain valleys of the Rocky Mountains; it is common among herders in the early spring; it is rarely fatal. The rash occurs first upon the legs. Typhus fever is not common in these valleys.

CEREBROSPINAL FEVER.

This is a meningitis with the symptoms of that condition; the rash occasionally might deceive. Tapping of the spinal canal will show a cloudy fluid rich in polymorphonuclear leukocytes which contain meningococci.

MALARIAL FEVER.

In its more severe type this condition might be confused with typhus fever, but the presence of the malarial organism in the blood will make the differentiation positive. Malarial fever does not occur because of overcrowding, but in those who have been exposed to the bite of the anopheles. In the rare cases of malarial infection which are seized with typhus fever, the subsequent course of the disease and its resistance to quinin administration will decide the diagnosis.

9. Dengue

(Breakbone Fever)

General Statements.—Dengue is an infectious disease occurring in tropical and sub-tropical climates, characterized by two sudden distinct febrile paroxysms, joint pains and irregular eruption. It is probably spread by the mosquito—Culex fatigans.

Characteristic Features.—The temperature slowly rises, sometimes reaching 105° F., remaining high for a few days—the average height being 103° F. At the same time there is severe pain in the head, eyeballs, back, extremities and joints. Every portion of the body is excruciatingly painful. The joints are *not usually painful to the touch and are seldom swollen.* There is a diffuse erythema over the body. On the third or fourth day the temperature falls by crisis and the patient seems almost entirely well. After the lapse of three or four days of comparative health, the fever and the muscle pains return, though not quite so severe, again accompanied by a rash over the entire body. This is a true eruption and not a mere erythema as was the first rash; it is not distinctive. It appears first on the palms of the hands and soles of the feet, occurring also on the lower extremities. Sometimes it appears in irregular coalescing patches showing areas of normal skin; in other instances the whole skin is red, as in scarlet fever. Different authors give a different name to its appearance; sometimes that resembling measles, sometimes rubella. The rash lasts about twenty-four hours and is followed by a branny desquamation. According to Guiteras, a diazo-reaction occurs in the urine. Von Berg has noted a leukopenia, the leukocytes being about one-half the normal number.

Conditions to be Differentiated from Dengue

In regions where it occurs the disease may be mistaken for:

Yellow fever
Rheumatic fever
Scarlet fever
Measles
Malarial fever.

YELLOW FEVER.

Yellow fever is a disease of one paroxysm. There is early jaundice and albuminuria; marked slowing of the pulse, also black vomit. Joint pains are not present; there is no characteristic rash, but in mild as well as severe cases a petechial eruption may occur on the face and chest. The mortality is high, while in dengue there are almost no deaths. Yellow fever is known to be spread by the Stegomyia fasciata, while dengue is probably spread by Culex fatigans. There is no Ehrlich diazo-reaction, and the blood count shows a normal number of leukocytes.

RHEUMATIC FEVER.

Rheumatic fever is more abrupt in the onset; the joints are red, swollen and extremely painful; there is often sore throat. The involvement of the various joints has its effect on the temperature. There may be an erythematous rash, but it occurs at varying stages of the disease, not being limited to the early stages. The heart often becomes diseased.

SCARLET FEVER.

Scarlet fever begins suddenly with high fever and sore throat. The rash, which is distinctive, begins on the trunk; there is glandular enlargement; a punctate rash with redness in the intervening spaces. The tongue becomes "strawberry" on the fourth or fifth day. Desquamation begins by large scales in the second week. There is leukocytosis.

MEASLES.

Measles is preceded by bronchitis and coryza; it must be borne in mind that Koplik spots are a characteristic symptom. Just before the rash the temperature falls, then rises; there is much conjunctivitis. The pains of measles are much less severe than in dengue. The chief symptoms in the former, as a rule, are pulmonary and not muscular.

MALARIAL FEVER.

Malarial fever is distinguished by distinct paroxysmal attacks, by intermittent or remittent fever and by the suddenness of the attack. The diagnosis can always be made by *examination of the blood* which shows the plasmodium.

10. Yellow Fever

Origin.—The causative factor is not known. It is an infectious disease. One of the most important life-saving facts of the century was established by Major Reed and his associates—Carroll, Agramonte, and Lazear—who proved beyond doubt that the disease is carried through the medium of the mosquito—Stegomyia fasciata—and not through fomites, water, or the air, as was for so long a time considered to be the case.

Symptoms.—The following description is copied from Carroll's article revised by McCrae, in "Modern Medicine," (1914 edition): "The earliest symptoms may be a premonitory feeling of malaise with pain in the back and loins, or slight frontal headache. There is usually a furred tongue, with constipation. In the majority of cases no marked premonitory symptoms are noted.

Onset.—The onset may be characterized by a sudden chill or rigor occurring at night or during the day. The patient may have lost his appetite and gone to bed to rest and sleep in the course of the day and awaken with high fever. Accompanying the fever there is sharp headache, generally frontal, but occasionally occipital. The face is flushed, the eyes injected, rachialgia is often present and the temperature may range from 102° to 106° F., or even higher. In mild cases the skin is moist, in severe cases dry and hot; constipation is usually present; the tongue is clean or highly coated and its tip and edges red. Photophobia may be pronounced or absent; the pains may be agonizingly severe or very mild in character; nausea may be absent, but usually it is present and followed by vomiting of a clear colorless or yellowish or greenish fluid. The symptoms are all more or less pronounced, depending upon the character of the attack. There may be great epigastric distress and the amount of urine passed may be small, but no albumin is found. At this stage the case resembles one of acute malaria, typhoid fever, or any severe infection.

Second Day.—On the second day the temperature may remain elevated or a fall may be observed; the headache and body pains may have abated or they may continue; the photophobia is more marked; flushing of the face and ocular injection are pronounced; a slight yellowish tinge of the eyeballs can be distinguished. The stomach may be quiet or the nausea or vomiting may be distressing but somewhat abated. There may be suppression or retention of urine. The skin of the chest is of a dusky hue, and upon pressure it is seen that the capillary circulation is sluggish.

Third Day.—On the third day the temperature may remain about the same or fall several degrees and then rise to near its original height, or it may fall gradually and decline during several days by lysis. A sudden drop of temperature to subnormal, with the appearance of black vomit and suppression of urine at this time, almost certainly means a fatal issue. More frequently after a brief decline on the second day, the temperature rises gradually to 103° or 104° F. and remains there with a morning fall of one or two degrees for one, two or three days, and then declines gradually to normal. Vomiting may return and the material ejected may contain fly-wing specks or coffee-ground granules, or it may consist of the brownish or brownish-black fluid known as black vomit.

Further Course of Disease.—The jaundice is now (fifth or sixth day) quite intense; great weakness and prostration are manifested. There may be cramping pains in the abdomen with the passage of a large amount of tarry material; these may be accompanied or followed by persistent hiccoughing, delirium, convulsions, coma and death. Remission or intermission in temperature may occur after twenty-four, forty-eight or seventy-two hours."

Conditions to be Differentiated from Yellow Fever

The diseases for which it may be mistaken are mentioned here, the distinguishing features of some of which will be given below:

Malarial fever
Blackwater fever
Dengue
Relapsing fever
Typhoid fever.

MALARIAL FEVER.

Malarial fever is easily distinguished by the examination of the blood, which will certainly establish the presence of a malarial parasite, a characteristic of the disease.

The symptoms of sudden fever with vomiting may be disturbing, but in malarial fever of the malignant type, there is, as a rule, tenderness over the spleen, and the jaundice is late.

BLACKWATER FEVER.

This always occurs in individuals who have had malaria, and such attacks can easily be established, particularly by the history, and by the fact that blackwater fever does not affect newcomers, as does yellow fever.

DENGUE.

Dengue is established by the fact that there are two paroxysms of fever, not one, as in yellow fever, and also by the facts that the pains affect the muscles and joints, as well as by the rash which is present first as an erythema and later a distinct rash. There is a decided leukopenia in dengue which is not present in yellow fever. There are two distinct rashes, erythema and a true eruption which does not occur in yellow fever.

RELAPSING FEVER—TYPHOID FEVER.

Relapsing fever and typhoid fever can be distinguished by blood examination—the blood of relapsing fever showing the spirochetes, and that of typhoid fever the Widal reaction and a culture positive for typhoid bacilli.

11. Epidemic Spinal Paralysis

(*Infantile Paralysis, Epidemic Poliomyelitis*)

Characteristic Features.—This disease, most frequent in the fall, is characterized by sudden fever, sore throat, temperature ranging to 103° or 104° F., and pains over the body and limbs. This acute condition lasts a day or two and is immediately followed by a flaccid paralysis of one or more of the limbs accompanied by great pain, simulating rheumatism or peripheral neuritis.

At times the constitutional symptoms are slight, the child may be put to bed apparently well; the paralysis is discovered the following morning. Occasionally the fever is high, there is marked delirium; almost immediate paralysis of the entire body, and death within twenty-four, forty-eight or seventy-two hours.

Organism.—The Rockefeller Institute, through Flexner, Lewis and Nogouchi, has proven that the disease is communicable from man to monkey and from one monkey to another. The organism has been discovered by Flexner and Nogouchi. According to these authors "the minute colonies are composed of globular or globoid bodies, averaging in young cultures from 0.15 to 0.3 of a micron in size. The bodies appear in a variety of arrangements; single, double, short chains, and masses. In older cultures certain bizarre forms have been noted. Monkeys have been inoculated with the twentieth generation of the culture; typical experimental poliomyelitis resulted."

Mode of Transmission.—The disease is probably communicated through the exudate from the nose and throat and gains entrance to the animal body through the same portals.

Pathology.—The final lesion is located in the anterior horns of the spinal column. At first there is a myelitis, a cerebritis, and a mild meningitis.

Symptoms.—The ordinary case of epidemic poliomyelitis is characterized by fever ranging from 100° to 103° F., a sore throat often mistaken for a follicular tonsillitis. The patient rather suddenly loses power in one or more of the limbs, the legs being more commonly affected. At the same time there is pain in the limbs, more especially in the paralyzed limb; there are no tendon reflexes in the paralyzed member. Retention of urine is common at first; leukocytosis is present. Lumbar puncture shows the spinal fluid to be under high tension. Many of the cases recover completely; in some there remains a weakness of one or more limbs and in others there is complete and permanent flaccid paralysis with wasting of the paralyzed limb and total loss of tendon reflexes. As stated above, some malignant cases die in a few hours of general paralysis. Other cases begin with a spinal paraplegia, the cord disturbance rapidly extending upward, resembling the type in Landry's paralysis.

Conditions to be Differentiated from Epidemic Spinal Paralysis

This disease has been mistaken for:
Hydrophobia
Rheumatism in the early stages

Ordinary acute infection of indefinite type
Meningitis
Multiple peripheral neuritis
Infantile cerebral paralysis
Rickets and scurvy
Bell's palsy.

In the first few hours it is impossible to distinguish acute infantile paralysis from one of the other acute infections. There is the same fever, the same general tenderness over the body, the same sore throat. With the onset of the paralysis, however, the diagnosis of course is positive for poliomyelitis.

Grave cases of the fulminating type have been observed by the author which simulated in every possible way an ordinary type of follicular tonsillitis, to be suddenly transformed into complete paralysis of the body, and death in forty-eight hours. One of these cases was at first looked upon as *hydrophobia*. There was not the laryngeal spasm—simply a paralysis of the muscles of deglutition which made the individual unable to swallow; there was no general convulsion, but a general flaccid paralysis.

Meningitis.

Meningitis differs from epidemic spinal paralysis in that there is rigidity of the limbs and neck, but it is closely simulated by the meningeal forms of poliomyelitis. Spinal puncture will show meningococci or other organisms in the fluid which are wanting in infantile paralysis.

Multiple Peripheral Neuritis.

This form of neuritis is characterized by the fact that the paralysis comes on gradually and by absence of fever; there is the history of chronic poisoning by some metal or drug, such as alcohol or lead, or by chronic infection from some hidden collection of pus, or it may follow some acute disease, notably diphtheria.

Infantile Cerebral Palsy.

In late stages the paralysis of cerebral palsy might be mistaken for that of infantile palsy, but there is spasticity, and a difference in reflexes—they are increased instead of being diminished as in spinal paralysis.

Rickets and Scurvy.

Rickets and scurvy are often accompanied by painful limbs and inability of the child to move these members, but the ordinary symptoms of the diseases are always present—the square head, bowed limbs and beading of the ribs in rickets, the spongy gums and swollen limbs of scurvy. The child resists any attempts to move the limb instead of having a flaccid paralysis.

BELL'S PALSY.

The loss of power in infantile paralysis may be principally marked in the distribution of the facial nerve—then the condition will resemble Bell's palsy. However, if a careful history and examination show that there has been fever, and that other muscles are involved, in addition to the muscles of the face, the diagnosis will be greatly influenced, as these are not symptomatic of Bell's palsy, this being characterized by a sudden loss of power in the muscles of the face without other symptoms.

12. Hydrophobia

(*Rabies*)

Etiology.—Hydrophobia is a transmissible disease caused by the bite of an animal suffering from rabies. It occurs in the following way: a healthy animal is bitten by a rabid animal; the animal contracts the disease and in turn can transmit it to another animal or to man. The disease is transmitted by the saliva—always *directly* transmitted; there is no carrier except the infected animal.

The virulent organism by which the disease is probably propagated is the Negri body which has very recently been cultivated by Noguchi; its life history is as yet unknown.

Hydrophobia is found in practically all of the lower animals and in man, but is most common in dogs and wolves.

Incubation Period.—The incubation period is very varied—from six weeks to as many months.

Symptoms.—The character of the wound and its situation have much to do with the morbidity of the bite. Bites on the hands and face which are apt to be lacerated and which receive the saliva straight from the fangs of the animal are most frequently followed by rabies.

Cases in which the wounded part is covered with clothing are less liable to be infected with the virus, because the part is less torn and the saliva is wiped from the fangs by the clothing. The toxic effect of the virus, whatever its character, has its first serious effect on the nervous tissue, because when the first symptom of the disease is shown the case is fatally ill. In this respect and in the fact that the disease can be prevented to a large extent by inoculation soon after the bite, it closely resembles tetanus.

Premonitory Stage.—The first symptom which appears is a feeling of depression. There is tingling and irritation at the site of the wound; this is followed by malaise, depression and melancholia, with an impending sense of danger. There then occurs difficulty in deglutition, especially in swallowing liquids. The patient is restless and suspicious, but for the first twenty-four hours he talks quite coherently. When the attempt to swallow liquids is made, the patient's actions are rapid and spasmodic; the liquid is gulped down rather than normally swallowed; the respiration is hurried and spasmodic; cutaneous hyperesthesia supervenes. At this time there is usually no difficulty in swallowing solids.

Later Stages.—Further along in the course of the disease liquids cannot be swallowed. The vessel is grasped by the patient, the liquid thrown into the mouth and at once there is a respiratory and laryngeal spasm, the liquid being spat forcibly from the mouth. Soon the respiratory spasms recur without being precipitated by an attempt at swallowing. These spasms are brought about by the least disturbance such as the pouring of liquids, bright light, and so on; they soon become almost constant. The patient becomes cyanosed and delirious; the mucous membrane is covered with thick mucus, constantly spat in the form of froth from the mouth, and frequently is expelled to some distance. The patient usually becomes relaxed before death and dies in coma.

According to Ravenel, a *paralytic form* of rabies occasionally occurs in man.

There is **never any attempt at biting or injuring an attendant.** The patient does not make noises that could be construed into the sound of a bark or growl.

Forms Found in Dogs.—Dogs develop two forms—the *furious* and the *paralytic* form.

Furious Form.—In the furious form the dog appears unwell and mopy for several days, then rather suddenly becomes sullen, snaps at its master, bites everything with which it comes in contact, froths at the mouth, the saliva flowing from its half open jaws. The eyes become bloodshot; the animal does not recognize any of its friends. When an attempt is made to drink water or other liquid the dog is often seized with convulsions. Sometimes the animal becomes paralyzed in all of its limbs before death occurs.

Paralytic Form.—The paralytic form is characterized first by paralysis of the muscles of deglutition and mastication. The dog then appears sick; its lower jaw hangs—the result of paralysis of the masseter muscles. Saliva dribbles from its wide open mouth and the dog walks quietly about attempting no harm to anything. In the course of twenty-four or forty-eight hours the limbs become paralyzed, the animal lies perfectly still and dies finally of exhaustion.

Diagnosis.—When an animal dies of hydrophobia the disease can be transmitted to other animals by subdural injections of an emulsion of the cord and especially of the higher nuclei of the brain. According to Ravenel ("Modern Medicine," vol. 1, 1914 edition):

> "It is by rapid staining of the plexiform nucleus of the pneumogastric nerve by which a diagnosis of the condition in a supposedly rabid dog can best be quickly made. If the dog has died of the disease and certain pathological changes are absent, it may be certain the dog did not have rabies. On the other hand if a dog has bitten a person it is necessary to discover as soon as possible whether the dog is rabid. Here a positive diagnosis must be made by searching for Negri bodies. These can be found early in the disease, and even after putrefaction changes have occurred. If the material is fixed in Zenker's fluid and stained with eosin and methylen blue, Negri bodies show red against the blue background." (Plate 2, Fig. 2.)

By the method of Williams an immediate diagnosis can be made by taking portions of the cerebral cortex about the crucial sulcus, the cortex of the cerebellum and the hippocampus major. This substance is crushed between two clean slides and a smear made by drawing them apart lengthwise, just as a smear is often made of sputum. These are dried in the air, fixed in methyl alcohol for three to five minutes and stained. According to Harris' method:

1. The smear is fixed one minute in methyl alcohol.
2. Wash in water and remove alcohol.
3. Immerse in old saturated solution of eosin in 95 per cent alcohol, one to three minutes (solution must be at least two months old).
4. Wash two or three seconds to remove excess of eosin.
5. Immerse in fresh solution Unna's alkalin methylen blue five to fifteen seconds.
6. Wash briefly in water.
7. Decolorize in 95 per cent alcohol, blot, dry in air. The entire process requires less than five minutes.

The nervous tissue shows certain changes and characteristic bodies, the Negri bodies.

Conditions to be Differentiated from Hydrophobia

The disease may be mistaken for:

Hysteria
Acute mania
Tetanus
Strychnin poisoning
Meningitis.

The most characteristic symptom of rabies is the *respiratory spasm;* the *inability to breathe accompanied by great cyanosis occurs in practically no other condition.* This fact influences diagnosis definitely.

HYSTERIA.

Hysteria may simulate rabies. The individual may have been bitten by a perfectly healthy dog and be in fear that rabies will develop. He becomes excited and many of the stigmata of hysteria are in evidence. He snaps, makes weird noises, but does not have the respiratory spasms which are characteristic of rabies. He may be made to talk in the midst of one of his assumed attacks. Patients never die as a result of an attack of hysteria. Recovery is a positive indication against hydrophobia.

ACUTE MANIA.

Acute mania may simulate hydrophobia, but it is characterized by the following special features: the mental symptoms are prominent; there is absence of the typical respiratory spasm; the death which occurs is longer delayed—an individual with hydrophobia dies in two or three days. In mania, also, there is no history of dog bite.

TETANUS.

Tetanus has almost always the history of an injury in which contamination with dirt has occurred, or the patient has had an injury with powder such as is used in making cheap fireworks.

The first symptom is a spastic condition of the jaw. There is no respiratory spasm apart from the general spastic convulsion which occurs; opisthotonos is a characteristic condition.

Negri bodies are not found in the tissues after death, as is the case in hydrophobia.

STRYCHNIN POISONING.

This condition always has the history of strychnin being taken by the mouth or hypodermic injection. There is no dog bite, no history of injury, no respiratory spasm, except as a part of the general convulsion, and no tenacious mucus appears about the mouth.

The convulsions are tonic, sudden and severe in character and the individual is entirely conscious. Death occurs after a very few hours.

MENINGITIS.

Meningitis is often sudden in onset, beginning with a convulsion, but the latter is clonic and general in character. There is no respiratory spasm, no history of dog bite—or at least no local signs—and no direct relation between this and the symptoms.

The writer has seen a pneumococcic meningitis with leukocytosis, with typical spinal fluid findings confirmed by autopsy where the patient had been bitten, and in the terror induced by the illness the case took on a form of lyssophobia, rather difficult to distinguish from true hydrophobia, except in this one particular—the respiratory symptoms were absent. This might easily occur where the meningitis was other than pneumococcic in origin.

13. Rheumatism

Organism.—This disease is infectious. The specific organism is still in doubt, but unquestionably belongs to the streptococcus group. Paine and Poynton have described an organism of this group, the diplococcus rheumaticus, which they have isolated from cases of rheumatism and which have been grown in susceptible animals and recovered from them.

Rheumatic Fever

Rheumatic fever is characterized by sudden, severe pain, swelling and redness of the joints, sometimes with exudate into or around the affected joints. The large joints are first affected, and the pain may, and frequently does, rapidly disappear from one joint to appear in another. Accompanying these symptoms there is often a chill, and always fever.

The fever is frequently distinctly intermittent, but it may run a course from 102° to 105° F. for a number of days, without intermission; or there may be a true hyperpyrexia. Accompanying this fever are often drenching sweats which leave the patient weak and exhausted.

In cases of rheumatic fever the joint is extremely tender, becoming red, swollen and tense. Often the tissues are so tense that the skin is red, bright and glistening. As has been stated, the arthritis almost always is polyarticular—one joint may be affected, then another, until almost every joint in the body has been diseased, usually the large joints first. With the subsidence of pain and swelling in a joint, the temperature often drops, to begin again with the infection of another joint. Thus days and even weeks may pass with this fever and pain recurring.

The disease is progressive; there are sweats; there is often inflammation of the pericardium or endocardium, causing pericarditis or endocarditis, varying from a mere irritation of the pericardium or valve, to a purulent or adhesive pericarditis or an ulcerative endocarditis, which may be lethal.

The end results of the true rheumatic inflammation of the endocardium or pericardium are those of chronic pericarditis or endocarditis, with their accompanying invalidism.

Similar Conditions.—Besides this severe and unmistakable rheumatic fever there is a series of other conditions which are certainly closely related to rheumatism, though the identity of the microörganism is not established, namely, chorea, multiple fibrous nodules, erythema nodosum and vague joint pains constantly called by the "laity" *growing pains*. All of these conditions are doubtless infective, and have in common with true rheumatic fever the fact that the *tonsils* are the portal of entry of the organism. They all may have inflammation of the joints, and endo- or pericarditis as part of their symptomatology.

The writer has notes of one case with the following train of symptoms: tonsillitis, multiple fibroid nodules, stiff and painful joints, chorea, endo- and pericarditis. The case with varying symptoms finally ended fatally from cardiac failure due to destructive endocarditis.

As important as any of these well-known forms are the vague joint pains, constantly neglected by the laity and by the physician. They are important, because of their great liability to be accompanied by endocarditis, which if the patient is not carefully examined, may cause lasting heart damage.

If the patient be examined in all these attacks, a heart lesion may be diagnosed and lasting harm possibly prevented by proper treatment. In true rheumatism the joints are not *permanently* disabled as they are in other conditions which simulate it.

Chorea

Chorea is unquestionably a member of the rheumatic group. It is characterized by irregular movements of the arms, legs and muscles of the face. The movements are entirely purposeless and made worse by excitement but can be temporarily controlled by the will. There is a tendency to endo- or pericarditis and changes in the mentality of the patient in certain instances.

The movements are jerky; occasionally the speech is interfered with, due to choreic movements of the tongue or lips. In certain instances there is a sort of delirium characterized by trifling manner or behavior; this may perhaps be due to the fact that the movements of the face, being constant, cause a sort of grimace which causes the patient to appear silly. Occasionally there is a true delirium due certainly to septic conditions accompanying these severe attacks. The movements are sometimes so slight that detection is difficult except by those in intimate relation with the patient, or they may be so severe that the patient must be restrained in order to prevent him from injuring himself or throwing himself out of bed.

In some instances only one side of the body is affected. In all cases, however, there is the same jerky purposeless character in all the movements. In the severe forms the attacks cannot be controlled by any effort of the patient. If joint complications or cardiac complications arise, there is fever, otherwise, the attacks are afebrile.

Conditions to be Differentiated from Chorea

HUNTINGTON'S CHOREA, ATHETOSIS, HABIT SPASMS.

Chorea must always be distinguished from Huntington's chorea and athetosis. The first occurs in middle-aged individuals. There is a history of heredity and the movements while jerky and purposeless are slower and more like athetosis. There is no tendency to joint pains or cardiac complications. Athetosis is rhythmic movements of the extremities occurring always when attempts are made to handle anything and frequently are independent of infection of any sort. It constantly accompanies a cerebral lesion.

Habit spasms—about the eyes, lips, nose, and the more severe ones of torticollis, often due to some local irritation—are to be distinguished from true chorea.

Erythema nodosum

Erythema, especially of the nodular type, is also a manifestation of rheumatic infection. Nodules occur under the skin, particularly upon the legs; they are hard, red, and extremely painful. Frequently they are accompanied by joint pains and heart complications, as are chorea and rheumatic fever.

Conditions to be Differentiated from Erythema nodosum

FURUNCULOSIS—SCARLET FEVER—SYPHILIS—MEASLES.

Erythema nodosum must be separated from a local lesion due to local infection of the skin, such as furunculosis. In furunculosis large inflammatory areas break down and form pus; erythema does not. The furuncles occur anywhere on the body; erythema is most frequent in the legs. Erythema is nodular in type and might be mistaken for scarlet fever, syphilis or measles. The absence of the constitutional symptoms of these diseases, together with joint pains or tonsillitis, will make the diagnosis.

Multiple Fibrous Nodules

Multiple fibrous nodules appear frequently as the first symptom of an attack of rheumatism. They are hard, elastic nodules, about the size of a pea, painless, and situated over the joints and along the tendon sheaths. They also are accompanied by the same joint and heart lesions as the true rheumatic fever.

Conditions to be Differentiated from Fibrous Nodules

HEBERDEN'S NODULES—GOUTY TOPHI.

Fibrous nodules can be mistaken for little else; they occur suddenly, are painless and are along the tendon sheaths. They differ from Heberden's nodules—these being over the articular ends of the phalanges of the fingers and toes.

Tophi of gout occur over articulations and in the pinna of the ear; they are deposits of mineral substances. There is often a history of gout.

Subacute pains, vague and fleeting, are simple painful conditions of the joints frequently accompanied by partial disability. As has been already stated the chief danger of this condition is the liability of cardiac effects, which may be entirely overlooked because not sought for.

Peliosis, rheumatic purpura, for want of better classification is still called by this name, although there is grave doubt as to the identity of rheumatism and peliosis. It is characterized by joint pains, at times with true involvement of joints and a sudden appearance of petechial hemorrhages, varying in size from a pin's head to a nickel. Frequently the hemorrhages and the joint involvement last over days or weeks. They are usually relieved by the salicylates.

Conditions to be Differentiated from Rheumatic Arthritis

Rheumatic fever, either acute or chronic, must be distinguished from the following conditions:

Arthritis deformans
Arthritis of syphilitic or gonorrheal origin
Traumatic arthritis
Septic arthritis
Gout
Acute osteomyelitis or periostitis
Tubercular arthritis
General arthritis
Scurvy
Osteomyelitis.

ARTHRITIS DEFORMANS.

It is unfortunately a fact that almost without exception cases of arthritis deformans are mistaken for rheumatism until serious joint lesions have occurred; this is especially the fact in the cases of spondylitis.

Arthritis deformans begins as a rule in the small joints of the fingers and hands—the large joints being involved later—is subacute in character and is unaccompanied by fever and heart complications. The affected joint is permanently lamed. The x-ray shows a rarefaction of the cartilages in early cases and later erosion of the same and true arthritic deposits. The joints are swollen, fixed and useless. The cartilages are destroyed and subluxations are apt to occur. In the hands there is the characteristic picture of ulnar subluxation. Finally all joints affected become fixed, and the patient falls into a state of complete helplessness. Marked atrophy of the muscles accompanies this joint involvement. This disease is slowly progressive; usually days, weeks or months pass, various joints being gradually involved, each exacerbation being followed by more disability in the affected joints. In acute cases it resembles rheumatism in its acute form—fever, sudden pain and swelling of many joints, large as well as small joints being involved—but the heart is not involved, and there is absolute disability of the joint.

Arthritis of Syphilitic or Gonorrheal Origin.

This condition is frequently mistaken for rheumatism. These affections are usually, although not always, monarticular. There is effusion into and around the joint. There is the history of syphilis or gonorrhea. They may both be accompanied by cardiac lesions. Cultures from the joints will show spirochetes or gonococci. A Wassermann complement fixation may be present in syphilis and also a complement fixation test in gonorrhea.

Traumatic Arthritis.

Traumatic arthritis is always the result of an accident and should not be mistaken for rheumatism. One traumatism to a joint constantly mistaken for rheumatism is subluxation of the ileosacral joint. If this condition is acute, the history of sudden disabling pain in the sacral region is unmistakable. When chronic, frequently there is the history of recurring attacks of pain and often a slightly movable joint can be demonstrated.

Septic Arthritis.

Arthritis due to measles and scarlet fever is not true rheumatism, but the arthritis is one result of the disease; it is probably due to the circulating toxin. This form is polyarticular and can always be diagnosed by knowledge of the previous disease. Care must be taken when one is confronted with a polyarticular arthritis, to carefully inquire as to the previous existence of a rash of measles or scarlet fever. The arthritis of streptococci is polyarticular.

Gout.

In the typical forms it suddenly attacks the phalangeal metatarsal joint of the great toe or the tarsal or metatarsal joints of the foot. The attack is sudden, there is great swelling and tenderness, sometimes the parts are much swollen and the tissues indurated. Usually there is a history of a gouty diathesis. In the chronic condition there is often a deposit of urates about the joint which is disabled. Tophi appear in the ears or about the articulations and are unmistakable.

ACUTE OSTEOMYELITIS OR PERIOSTITIS.

Nothing is more distressing than the mistake of considering acute osteomyelitis or periostitis as rheumatic in origin. In these conditions there is pitting on pressure over the affected area, other portions of the bone than the joint are affected, and the lesion is localized. It never affects numerous joints. There is always a marked leukocytosis, more marked than in rheumatism.

TUBERCULAR ARTHRITIS.

Tubercular arthritis is so frequently considered rheumatism, that valuable time is lost before the true nature of the condition is recognized. In tuberculosis, whatever joint is affected, there is almost always complete or partial immobility; there is fever, and unless suppuration has occurred, there is no leukocytosis. The joint is enlarged; as a rule there is but little redness. Arthritis of the knee or hip should never be considered rheumatism, until the possibility of tuberculosis is excluded. The use of the tuberculin test, by injection or von Pirquet, should help settle the question.

In all the various forms of arthritis it must be remembered that the *cause must be sought for.* If a joint is deformed after inflammation the inflammation was not due to rheumatism. As time passes the cause for many chronic cases of arthritis, formerly called rheumatism, is more and more frequently found in diseased nasal sinuses, in unsuspected inflamed roots of teeth, in inflamed tonsils, in the prostate gland, in the urethra, etc. Careful examination of all portions of the body must be made in order to make a true diagnosis of the cause.

14. Acute Tonsillitis

(*Follicular Tonsillitis, Lacunar Tonsillitis*)

Initial Symptoms.—This condition is ushered in with severe headache, backache, fever—often rising to 104° F.—sore throat and general depression. The tonsils are red and swollen and the follicles are filled with yellowish plugs. These plugs are usually scattered over the tonsils, and located in the follicle, but may cover the whole tonsil with a pultaceous mass. Sometimes there is so much swelling that swallowing is almost impossible. The cases are infectious.

Organism.—The organisms found are extremely varied; streptococcus and staphylococcus are in the majority.

Exudate.—One familiar with throat conditions in general practice can usually distinguish the exudate common to tonsillitis from that due to diphtheria, but an absolute diagnosis cannot be made without throat culture and examination.

The characteristics of the exudate of acute follicular tonsillitis are: the exudate is in the follicles; it may become a pultaceous mass, it is easily removed and in this case leaves a clear surface which does not bleed by reason of the removal of the exudate. However, if the tonsil is much disturbed or roughly handled, it will bleed.

If the exudate is due to a streptococcus infection there may be a true membrane not beginning in the follicles which adhere closely to the underlying mucous membrane, and its removal causes the surface to bleed. It closely resembles diphtheria. In case of doubt, especially in very mild cases, a smear, or better, a culture, of the throat exudate should be made. Jacobi long since said that more diphtheria stalked about under the guise of tonsillitis than was diagnosed. It is ambulant cases of diphtheria to which an epidemic often owes its spread.

Every practitioner should make his own cultures, or have them made for him in any case of the least doubt. I would insist that there are no symptoms or physical signs, save cultural methods, which will always distinguish between a staphylococcic or streptococcic sore throat and diphtheria (Plate 1, Fig. 2).

Diseases to be Differentiated from Acute Tonsillitis

Diphtheria
Scarlet fever
Tonsillar or peritonsillar abscess
Postpharyngeal abscess
Vincent's angina
Syphilitic sore throat.

DIPHTHERIA.

Diphtheria may have as severe and rapid a beginning as tonsillitis, but usually the onset is slower. There is a stiffness of the throat, rather than severe soreness. The exudate is a membrane which adheres to the *underlying mucous membrane and causes bleeding when it is removed.*

Sometimes it is confined solely to the tonsils. Usually it affects other surfaces, the uvula and the pharyngeal being the most commonly affected.

Diphtheria bacilli can always be demonstrated by careful cultural methods. The glands at the angle of the jaw are apt to become much swollen. The condition is often followed by severe complications—suppuration of the glands, paralysis, or often severe toxemia and death.

Acute tonsillitis rarely kills directly, its sequelae, such as endocarditis, may.

Scarlet Fever.

Scarlet fever begins abruptly within twenty-four hours. There is the typical scarlet rash beginning as a rule on the chest. The throat becomes brick red with some exudate, usually stringy in character; rather, the throat looks red, the tonsils swollen and covered irregularly in spots with an exudate.

Diphtheria bacilli, except as an indication of intercurrent disease, are not present. Gangrene of the throat may occur; ear complications are common; there is desquamation of the skin, making its appearance eight to ten days after the beginning of the disease.

Tonsillar or Peritonsillar Abscess.

Tonsillar or peritonsillar abscess or quinsy may begin with all the characteristics of a follicular tonsillitis, or it may begin in an inflammation; however, in two or three days the tonsils enlarge, the whole fauces become swollen and often edematous, the glands at the angle of jaw become hard and indurated. Swallowing becomes practically impossible, liquids being regurgitated through the nose rather than swallowed; finally, after several days, an abscess points usually in the soft parts just above the tonsil. These signs are not present in follicular tonsillitis.

Postpharyngeal Abscess.

Pharyngeal abscess is lacking in any primary involvement of the tonsil, either follicular or interstitial; instead there is difficulty of swallowing. A swelling of the posterior pharynx is noticed; the tissues become red, tense and often edematous; palpation of the posterior pharyngeal wall will show a fluctuating mass in that position.

Vincent's Angina.

Vincent's angina is characterized by small punched-out ulcers in the tonsils, and also by the fact that the exudate is cheesy in character and has an extremely disagreeable odor. Smears from the tonsillar exudate will show numerous bacilli fusiformis and spirochetes.

Syphilitic Sore Throat.

Syphilitic sore throat is distinguished by general glandular involvement, by the appearance of a skin rash of syphilis, by the presence or history of an initial lesion, and by the presence of a Wassermann reaction.

15. Acute Catarrhal Fever

(*Acute Coryza, Common Cold*)

Origin.—This condition is due to an infection of the upper air passages by various organisms, none of which have as yet been proven specific, although the Micrococcus catarrhalis is frequently present.

The Micrococcus catarrhalis is easily cultivated from the mucous discharges of the throat and nose. The disease is unquestionably infectious, constantly attacking members of the entire household.

Symptoms.—The patient is dull, has headache, is chilly, sneezes constantly; there is pain in his limbs, and sometimes he has a sore throat.

The sense of smell is often seriously interfered with; the voice is husky, due to a laryngitis. At times, accompanying this condition, there is inflammation of the middle ear, frequently resulting in suppuration.

Conditions to be Differentiated from Acute Catarrhal Fever

This state may be mistaken for:

Influenza—also for Measles.

INFLUENZA.

Acute catarrhal fever is constantly diagnosed as influenza—indeed the symptoms almost exactly resemble mild cases of influenza. A differentiation must really depend upon the presence of influenza as an epidemic, and the isolation of the bacillus of influenza from the discharge.

MEASLES.

Acute catarrhal fever is also constantly mistaken for nasopharyngeal catarrh, which precedes measles. From this it is easily distinguished by the presence of Koplik's spots on the first or second day of measles, and the appearance of a rash of measles on the third or fourth day.

16. Febricula

(*Ephemeral Fever*)

This is a short, continued fever depending upon one of many causes.

Whether the disease really deserves a special heading is extremely doubtful, as certainly the fever is the result of some cause, and we must limit the name febricula or ephemeral fever to a fever of very short course, where the infection is entirely unknown.

Symptoms.—It is characterized by rise of temperature lasting three or four days, without any local causes which would explain the fever,

Conditions to be Differentiated from Febricula

It must be carefully differentiated from the beginning of TUBERCULOSIS, from TYPHOID FEVER, from any of the infectious diseases, or from FEVER AS THE RESULT OF SOME GASTRO-INTESTINAL DISTURBANCE. Only the absence of a localized foci of infection as the result of constant, frequent, careful examination, will justify a diagnosis of febricula.

17. Infectious Jaundice

(*Weil's Disease, Epidemic Catarrhal Jaundice*)

Origin.—This is an infectious disease due to an unknown organism, which brings about a severe catarrhal jaundice with toxic symptoms.

Geographical Distribution.—The disease has occurred in various parts of the United States, and also in Europe, Asia, and Africa.

Symptoms.—The symptoms are first, vomiting, epigastric distress, fever, headache, pains in the back, always accompanied by jaundice, enlarged liver and spleen, frequently nephritis, and the usual nervous symptoms of a severe infection. A typhoid state occasionally occurs.

Conditions to be Differentiated from Infectious Jaundice

It must be distinguished from the following conditions:

Simple catarrhal jaundice
Yellow fever—Dengue
Pneumonia
Malignant icterus—Acute yellow atrophy
Cholelithiasis
Acute chronic cholangitis—Errors of diet.

Simple Catarrhal Jaundice.

Simple catarrhal jaundice is never epidemic; it is traceable to a cause, such as overeating. It is easily diagnosed by the fact that Weil's disease is evidently infectious and is epidemic, whereas the cases of catarrhal jaundice are isolated, and the cause can easily be discovered.

Yellow Fever—Dengue.

In districts where yellow fever and dengue abound, infective jaundice might be mistaken for them. However, in dengue there is a characteristic two-cycle fever, often erythema, and marked joint and muscle pains.

Yellow fever is perhaps more difficult of diagnosis, but the black vomit, the depression, the fatal character of many cases, and above all the history of infection through the stegomyia makes the diagnosis certain.

PNEUMONIA.

A severe jaundice, hemolytic in character, sometimes accompanies an infection like pneumonia. A blood culture and discovery of a local lesion in the lung will make the diagnosis.

MALIGNANT ICTERUS—ACUTE YELLOW ATROPHY.

Malignant icterus and acute yellow atrophy, which are perhaps only grave forms of infection of the bile passages, can finally be diagnosed by the fact that the cases do not occur in groups and are not transmitted from one individual to another.

CHOLELITHIASIS.

This condition is not infectious. There is a previous history of preceding indigestion; there are attacks of pain referred to the gall-bladder region; during or after an attack there is tenderness in this region. Jaundice may be absent or present. Local inflammatory complications are often present.

CHRONIC CHOLANGITIS.

Chronic cholangitis is characterized by jaundice, usually with fever of intermittent type, with leukocytosis. There is usually tenderness in the epigastrium. The condition is not infectious.

ERRORS OF DIET.

These have vomiting and epigastric distress as symptoms; there may be jaundice. The condition is not infectious; there is no fever.

18. Milk Sickness

(*Trembles*)

This curious disease occurs in the western part of the United States; so far as is known, it has never been reported east of the Allegheny Mountains.

Cause.—It is caused by eating the flesh or drinking the milk of diseased animals; butter and cheese made from the milk of these animals are also poisonous. The animals themselves are subject to trembles.

Lately (1908) Harrison and Jordan have demonstrated a bacillus lactomorbia, from cultures of which the disease may be caused in animals.

Symptoms.—The symptoms are those of acute intoxication—vomiting, fever, thirst, a particularly foul breath, and swollen and tremulous tongue; in severe cases irritation, restlessness, convulsions, often ending in death.

Diagnosis.—Milk sickness can be diagnosed by the two following means only: by tracing the disease to meat or milk of animals infected with trembles, or by cultivating the suspected organism; if found to be the bacillus lactomorbia, the character of the disease is proven.

Conditions to be Differentiated from Milk Sickness

The condition might be mistaken for:

Acute enteritis with fever
Typhoid fever.

ACUTE ENTERITIS.

In acute enteritis the absence of a history of contact with animals or animal products affected with trembles will make the differentiation.

TYPHOID FEVER.

Typhoid fever has been mistaken for milk sickness, but the Widal reaction, blood culture, rose spots, the temperature curve, and the signs characteristic of typhoid, together with the absence of contamination by affected animals, will enable one to come to a definite conclusion.

19. Glandular Fever

Glandular fever is an acute condition occurring frequently in children. It is believed to be a specific fever and not a simple acute adenitis.

Symptoms.—It is characterized by sore throat, irregular fever and swelling of the lymph glands, particularly of the neck, though glands over the entire body may be involved.

The symptoms are accompanied by pain in the neck, swollen tonsils, nausea and vomiting. The liver and spleen are frequently enlarged. On the third or fourth day the posterior cervical glands enlarge and become painful. There is occasionally a cough, apparently due to enlarged peribronchial or peritracheal glands.

Complications.—There are very few complications but among them may be suppuration of the glands and nephritis.

Conditions to be Differentiated from Glandular Fever

This disease must be separated from:

Tubercular adenitis
Adenitis due to some local cause
Hodgkin's disease
Acute leukemia
Syphilis.

TUBERCULAR ADENITIS.

Tubercular adenitis is chronic in character; the glands have a tendency to suppurate more than those of glandular fever, they are more likely to be unilateral. There are not the same symptoms of an acute infection.

ADENITIS DUE TO SOME LOCAL CAUSE.

Adenitis due to a simple local infection of the mouth, such as stomatitis, carious teeth and other mouth diseases, is not apt to be so frequently accompanied by general symptoms as is glandular fever.

Adenitis due to scarlet fever, diphtheria, or measles, have accompanying symptoms of these diseases.

HODGKIN'S DISEASE.

This disease is chronic. The glandular masses are larger; neither is there the acute fever of glandular fever. Instead the fever is long continued in remittent paroxysms. Examination of the blood is helpful.

ACUTE LEUKEMIA.

Acute leukemia is characterized by marked leukocytosis, either composed of lymphocytes or polymorphous leukocytes.

SYPHILIS.

Syphilis has other symptoms than those of adenitis—rash, sore throat, alopecia, and lastly the Wassermann reaction.

20. Miliary Fever

(*Sweating Sickness*)

Miliary fever, or sweating sickness, is a disease which is characterized by sweating, fever and eruptions of the miliary vesicles. Cases are rare at present, but according to authorities, an epidemic of some extent occurred in France in 1837. There are also reports of an outbreak in Austria. A characteristic feature of the disease is that a large number of persons are attacked at one time.

Symptoms.—The onset is sudden; there is sweating as the first symptom; fever occurs, often rising to 103° F., sometimes higher. There is rapid pulse and much palpitation of the heart.

Nervous phenomena are common; there are prostration, delirium, convulsions and laryngeal constriction. Hemorrhages may occur from the nose and other mucous membranes.

Sweating is a characteristic symptom; it begins with the fever, continues during its course and is greatest when the fever is highest.

An eruption occurs on the third or fourth day. A general erythema is present, in addition to which appear (*a*) sudamina (miliaria alba or crystillina), (*b*) red papules becoming vesicular (miliaria rubra), (*c*) petechiae of variable size (purpura miliari [Boggs]).

With the appearance of the rash, the fever and the nervous symptoms gradually disappear. As a rule cases recover, but occasionally the disease is so severe that the patient dies within a few hours.

Differentiation.—MEASLES may be mistaken for the disease, but the presence of Koplik's spots, severe catarrhal symptoms and the absence of the characteristic sweat in measles, will distinguish the two diseases.

21. Foot and Mouth Disease

Foot and mouth disease is a condition which is contracted from cattle, sheep and pigs. It spreads among cattle very rapidly, thence to man.

Symptoms.—It is characterized in man by fever, usually moderate, though, according to Boggs, ranging sometimes as high as 104° F.

The case usually begins with an infection of the mouth. At first this becomes hot and dry; vesicles appear on the edges of the tongue, on the lips and cheeks, having been preceded by a hyperemia of these parts. The vesicles are at first discrete, but they rapidly become confluent; they extend in severe cases to the pharynx and even into the esophagus. Sometimes there are blebs on the skin about the nose and lips which in very severe cases may spread over the whole body. The fever usually falls with the appearance of the vesicles. As a rule the case is relatively short in duration, depending upon the spread of the vesicles and the severity of the infection.

Conditions to be Differentiated from Foot and Mouth Disease

The condition may be confused with any of the milder forms of stomatitis:

Aphthous stomatitis
Ulcerative stomatitis.

APHTHOUS STOMATITIS.

Aphthous stomatitis occurs most frequently in children, and while there is marked lassitude, inability to eat and fever, the necessary history of infection by animals is entirely wanting.

ULCERATIVE STOMATITIS.

Ulcerative stomatitis is often followed by destruction of the tissues beneath the vesicles.

There is without exception in foot and mouth disease a history of intimate connection with animals which are infected. In cases of doubt, inoculation of material from the vesicles will influence a diagnosis.

22. Rocky Mountain Spotted Fever

Geographical Distribution.—Rocky Mountain spotted fever is an acute infectious fever common in the valleys of the Rocky Mountains, in Idaho, Montana and along the valleys as far as Mexico.

Characteristic Features.—This disease is characterized by fever, headache, muscular pains and a petechial rash.

Etiology.—We owe our knowledge of this disease principally to Maxey and to Wilson and Channing, but also to Ricketts, who discovered the clinical course of the fever and its method of dissemination.

The actual cause of the disease is not known. King and Ricketts have proven that it is spread through the medium of a tick, the Dermacentor occidentalis. These authors have transmitted the disease to animals by means of bites of these ticks; they also found that the ova and young of the ticks contain the infective material:

> "A certain percentage of the female ticks which have acquired the disease as a consequence of feeding on animals—the latter having been infected by other ticks—transmit the disease to their offspring through the egg. The new generation during the process of feeding, transfer the virus to certain of the susceptible small wild animals (ground squirrel, rock squirrel, chipmunks, ground hogs, and perhaps others), and this may take place either during the larval, nymphal or adult stage, hence at various times of the year. During the infection of the wild animal it is required that hitherto normal ticks either as larvae, nymphs or adults, acquire the disease by feeding simultaneously with, or shortly after, the feeding of the infected ticks."

Predisposing Factors.—Apparently the type of the disease varies in severity in the various areas where it occurs. The disease occurs in the early spring months, just after the snow has melted, and is most common among males for the reason that they, particularly herders, are led by their occupation to be in the meadow lands and grasses at this time of the year, and are bitten by the tick.

Symptoms.—There is a short period of malaise, followed usually by a well-marked chill; these chills may be repeated throughout the course of the attack.

At the beginning there is severe aching of the bones and muscles, pains in the joints, and severe headache. Constipation is the rule. The skin is dry, the tongue is coated, sordes appear early and the case in the beginning has the appearance, except for the chill, of typhoid fever.

Temperature.—The temperature rather rapidly develops and reaches 102° or 103° F. on the third or fourth day; it may go much higher, reaching even 107° F. There is usually a slight evening increase and morning decrease. On recovery, the temperature falls by lysis.

Skin.—The most characteristic feature of the disease is the skin eruption. From the second to the fifth day after the chill a macular rash, which rapidly takes on the characteristic petechial appearance, is seen around the ankles and upon the wrists; it then extends over the entire body. Occasionally the rash will cover the entire body in twelve hours; more usually, however, it takes a longer time. A desquamation occurs in about the second week of the disease, which is best seen on the soles of the feet and the palms of the hands. Occasionally there is jaundice.

Severe Cases.—In severe cases the patients become delirious and pass into a typhoid state. There is no sign of meningitis.

Pulse.—The pulse is rapid—out of proportion to the temperature.

Blood.—The blood is only slightly changed, the red cells being normal, with white cells being from twelve to thirteen thousand.

Digestion.—Except for constipation, there is no unusual sign of disturbance of digestion.

Urine.—The urine frequently shows albumin and tube casts.

Conditions to be Differentiated from Rocky Mountain Spotted Fever

The condition must be separated from:

Typhoid fever
Typhus fever
Cerebrospinal meningitis.

TYPHOID FEVER.

Typhoid fever is marked by the slow onset, typical abdominal rash and long course, by diarrhea, the Widal reaction and blood culture. There is no history of exposure to ticks.

TYPHUS FEVER.

Typhus fever is not found in the Rocky Mountains, nor does Rocky Mountain fever occur when typhus abounds. The latter is a disease of filth and crowding, distributed by the body louse, in which the decline of fever is by crisis—not by lysis—as in Rocky Mountain fever.

CEREBROSPINAL MENINGITIS.

Cerebrospinal meningitis is charcterized by stiff neck, convulsions, herpes and occasional petechiae. The spinal fluid contains polymorphonuclear cells, crowded with meningococci. All these symptoms are lacking in Rocky Mountain spotted fever.

23. Swine Fever

Swine fever is contracted by making post mortems on hogs infected by the disease. There is swelling, with a bluish tinge to the fingers of the affected hand. The lymph glands are frequently enlarged and there is painful erythema which creeps from one place to another.

Diagnosis.—The causative factor here is the diagnostic one. This infection can be distinguished from other septic conditions by the fact that the causative agent was obtained directly from the hog.

24. Rat-bite Fever

Cause.—This disease is caused by a rat-bite. It is infectious. Several organisms have been described as the cause, though none actually have been proven to be so.

Course of Disease.—The wound usually heals quickly; later it becomes swollen, red, and eroded. "An ulcer forms, and neighboring lymph glands are involved." There is a chill, and a fever which lasts three or four days—the temperature may rise to 104° F. There is muscular pain, a skin rash which resembles measles, and a moderate leukocytosis. Finally the temperature falls, and the patient feels quite well. There is no enlargement of the liver and spleen. These symptoms occur on the average two weeks after the reception of the bite, though they may occur several months after it.

Later Stages.—This feeling of well-being lasts for one or two weeks when a second paroxysm occurs of somewhat shorter duration; a third or fourth may occur, each successive paroxysm lasting a shorter time. Sometimes they recur after an interval of one year—they are usually of very short duration.

Conditions to be Differentiated from Rat-bite Fever

The disease might be taken for:
Measles
Malarial fever.

MEASLES.

Measles is differentiated by its characteristic sign of mucous membrane inflammation—Koplik's spots—and further, by the fact that there is no history of rat-bite.

MALARIAL FEVER.

Malarial fever has blood picture entirely different from that of rat-bite fever. In the former the spleen is enlarged; there is a history of exposure to anopheles, but no history of rat-bite.

25. Psittacosis

This is a disease of birds—especially parrots—which is transmitted to man. *In birds* there is loss of appetite; enteritis and general debility are common. When parrots have been nourished by mouth to mouth feeding, the disease is most easily communicated. There is a diphtheritic stomatitis, with enlargement of the submaxillary glands.

In man, according to Boggs, the disease resembles typhoid fever complicated by pneumonia. Sometimes the onset is gradual, sometimes sudden, with headache, malaise, photophobia, loss of appetite, nausea, constipation and weakness.

The rise of temperature is rather rapid, 102° to 104° F. being attained. There is mucopurulent sputum which is sometimes bloody; fine moist râles are heard throughout the lungs. With pneumonic symptoms there are areas of consolidation.

The duration of the disease is from five to fifteen days.

Conditions to be Differentiated from Psittacosis

It may be mistaken for:

Typhoid fever
Pneumonia
Paratyphoid fever
Influenza,

but in psittacosis there is always the history of infection through the medium of birds.

TYPHOID FEVER.

The agglutination reaction present in typhoid is absent in cases of psittacosis; also any strain of colon bacillus. The characteristic blood changes of typhoid fever are present.

PNEUMONIA.

There is not the same leukocytosis in psittacosis as there is in pneumonia, and the lung condition is a typical consolidation which is not present in psittacosis.

INFLUENZA.

Influenza is distinguished by the presence of typical respiratory symptoms which are absent in psittacosis. The influenza bacillus is present in the mucous discharges.

Section II

Diseases Due to Physical Agents

1. Sunstroke—Heat Exhaustion—Muscular Spasm

Sunstroke

Causes.—Sunstroke is a condition which follows exposure either to direct heat of the sun or to artificial heat.

Symptoms.—The patient is suddenly stricken. There are a few premonitory symptoms, as headache and dizziness, but these are usually disregarded by the patient who is accustomed to working in the heat. With or without these premonitory symptoms, the patient falls and becomes unconscious; the temperature is found to range from 105° to 110° F. There is dyspnea, rapid heart action, often cyanosis, and at times convulsions; the skin feels extremely hot.

Conditions to be Differentiated from Sunstroke

This set of symptoms as occurring from direct exposure to heat can be mistaken for:

Malignant malarial fever
Uremia
Diabetic coma
Apoplexy.

MALIGNANT MALARIAL FEVER.

The symptoms of malignant malarial fever may suddenly occur while a patient is exposed to high temperature. The patient will be unconscious, cyanosed, and have high fever. Malarial fever can be differentiated in the first stages by examination of the blood; malarial plasmodia will be found, which are absent in sunstroke.

UREMIA AND DIABETIC COMA.

Uremia and diabetic coma may also make their appearance suddenly while a patient is exposed to the heat. High blood pressure in uremia, however, and the curious deep respiration in diabetes, will suggest the diagnosis. In uremia there is high blood pressure, and often complete anuria. The urine in uremia will contain albumin and tube casts, in diabetes, sugar and ketone bodies. It is of course possible for persons with albumin and tube casts or sugar in their urine to develop sunstroke. The temperature of both anemia and diabetes is at or about normal.

APOPLEXY.

Apoplexy may occur under the same conditions which develop sunstroke, but the absence of fever and the presence of a suddenly occurring paralysis will point to apoplexy. In the latter, a spinal puncture will show a bloody fluid, in a large number of cases; the fluid in sunstroke is clear.

Heat Exhaustion

Causes.—Heat exhaustion is a condition resembling syncope, and occurring in persons who are exposed to high temperatures, *whether the heat is the result of the direct rays of the sun, or an artificial heat,* such as is common in some of the industrial plants—particularly in iron manufacturing plants. It is superinduced by poor physical condition of the patient and by alcoholism.

Symptoms.—The patient is weak, has a tendency to faint, the body temperature is low, the skin cold and clammy.

Conditions to be Differentiated from Heat Exhaustion

It must be differentiated from the *same set of symptoms occurring without the influence of heat.* Here the same diagnostic methods for APOPLEXY, MALARIA and UREMIA must be applied as in the case of sunstroke.

Muscular Spasm

(*Myospasm*)

Muscular spasm occurring in workers before furnaces and stokers in ships was first described by Edsall, in 1904. The condition was well known to those practicing among mill workers and stokers before Edsall's paper, but the communication was, so far as the writer knows, the first to actually describe the condition.

Symptoms.—The patient, after he has been working in this intense heat for a greater or shorter time, is suddenly seized with extremely severe spasms and pain in the muscles of the arms, legs and abdomen; the spasms are tonic in character and come on in paroxysms. In one case seen by the writer, the muscle spasm was so great that the humerus was fractured when a bystander took hold of the arm to restrain it.

Conditions to be Differentiated from Muscular Spasm

This condition may be confused with:

Hysteria
Spasms due to some acute or chronic brain lesion
Tetany
Some acute abdominal condition.

HYSTERIA.

Hysteria may be excluded by the fact that the contractions are so manifestly independent of the will and are so painful; also by the history of the patient's exposure to heat before the time of examination.

SPASMS DUE TO SOME ACUTE OR CHRONIC BRAIN LESION.

Brain lesions are differentiated by the history of the case, by the painless results of brain lesions, and by the fact that spasms due to brain lesions rarely, if ever, are bilateral—as are spasms due to heat.

TETANY.

Tetany can be excluded by the absence of Chvostek's and Trusseau's signs and by the history of the case.

ACUTE ABDOMINAL INFLAMMATION.

Pancreatitis, appendicitis or perforations may be differentiated by the presence of shock, of abdominal tenderness, and by the absence of terrific paroxysmal contractions and leg and arm spasms—common in spasms due to heat.

2. Caisson Disease

(*Compressed Air Disease, Diver's Paralysis*)

Etiology.—This is a condition affecting the workers in caissons or divers when they are decompressed rapidly, after being submitted to high pressure in their work.

A caisson is a steel tube open at the bottom and closed at the top, with a door which leads into a decompressing or air chamber. The workman enters the air chamber, closes the door and equalizes the pressure in the air chamber with that in the caisson. He then enters the caisson. In returning he reverses the process by a set of valves. The man works in the caisson proper under a pressure of air greater than the pressure in the surrounding water. When the workman is in the atmosphere of high pressure, the tissues of the body are saturated with nitrogen; as he comes into an atmosphere of lower pressure or is decompressed, the nitrogen is given off in bubbles in the liquids of the body—particularly in the blood.

The symptoms which arise are due to the bubbles of nitrogen causing emboli in the various tissues. The more adipose an individual, the more likely is he to develop symptoms. The slower the decompression, the less likelihood of symptoms.

Symptoms.—The symptoms, as described by Andrew Smith in Allbutt's "System of Medicine," are as follows:

> "In the order of frequency—pain, often very severe, in one or more of the extremities, and occasionally in the trunk; pain in the epigastrium, which may or may not be associated with nausea and vomiting; paralysis, more or less extensive and complete; headache, vertigo, and coma. In rare cases sudden death occurs almost without preceding symptoms. The pain, which is of a neuralgic character, may be slight and transient, or extremely severe and persistent. It is usually intermittent or remittent. It may come on gradually, increasing in severity until it becomes absolutely intolerable; or it may begin at once in its full intensity. The knees, legs and hips are most frequently attacked, but the arms or trunk may be the first to suffer. Sometimes the greatest suffering is in the back, and particularly in the lumbar region. Epigastric pain is frequent; if not quickly relieved, it is followed by sickness and vomiting. Vomiting may take place without preceding gastric pain, and then is usually accompanied by giddiness or other evidence of cerebral origin.
>
> Paralysis occurs with increasing frequency and completeness in proportion to the degree of pressure and the duration of the exposure to its influence. The lower portion of the body is more liable to attack but the upper extremities are not exempt. The paralysis is of sensation as well as of motion, but it gives no relief to the pain. The part is insensible to pinching or to the prick of a pin, while at the same time it is the seat of extreme suffering. But there is no necessary relation between the pain and the paralysis, as either may occur separately. The paralysis varies in degree, from a slight and transient paresis with some impairment of sensation to complete and permanent loss of motion and sensation in the affected part. Even the minor degrees usually include the bladder.
>
> Symptoms indicating cerebral disturbance of a transient character are often observed, such as headache, double vision, giddiness, incoherence of speech, and occasionally syncope. The skin is often mottled in patches, some of which are veritable ecchymoses; others are the result of stasis in the distended capillaries, and can be rubbed away by persistent friction.
>
> The duration of an attack varies extremely. It may last a few hours, or it may continue for six or eight days. Paralysis may be recovered from in a few days, or it may be protracted for weeks or months. Death occurs only in cases that are severe from the first; and, except when due to secondary lesions, it usually takes place shortly after the attack."

Conditions to be Differentiated from Caisson Disease

This is one of the conditions in which *a history* is essential for a diagnosis.

The condition can scarcely be mistaken for anything else. With a history of an individual working in a caisson or in a diver's suit, the only possible condition which it could simulate would be uremia or paralysis due to rupture of a blood vessel.

UREMIA.

Uremia can be discovered by the condition of the urine, heart and blood pressure. A mistake is unlikely to occur if careful examinations are made of individuals before they are compressed.

PARALYSIS DUE TO RUPTURE OF A BLOOD VESSEL.

Hemorrhages into the brain tissues are characterized by hemiplegia, by partial or complete unconsciousness and by loss of speech. The spinal fluid is apt to be bloody.

3. Mountain Sickness

Cause.—This is a condition due to a cause which is in direct contrast to that of caisson disease: the patient is made ill by an atmospheric pressure below normal, such as is found in high altitudes. The symptoms are all the direct result of a lack of oxygen in the lungs.

Symptoms.—The symptoms are ringing in the ears, dyspnea, a tendency to syncope, dizziness and nausea.

Conditions to be Differentiated from Mountain Sickness

The only conditions with which these symptoms can be confused are:

HEART AND LUNG CONDITIONS WHICH HAVE THE SAME SYMPTOMS IN NORMAL ATMOSPHERIC PRESSURES.

They may be distinguished by careful examination of the heart and lungs. If these organs are normal, the symptoms may be attributed wholly to the unusual pressure. If the organs are at fault, the symptoms may simply be increased by the heart condition found. Except by getting the patient in a lower altitude, there is no way in which an accurate differentiation can be made.

Section III

The Intoxications

1. Alcoholism

Forms.—Alcoholism may be conveniently divided into acute and chronic forms.

(*a*) *Acute Alcoholism*

Acute alcoholism is the result of an overdose of alcohol taken in a short interval of time, causing the symptoms of intoxication by that drug. The symptoms, unfortunately too well known, may be described first as exhilaration, followed by incoördination of the muscles with decided mental disturbances, and finally, unconsciousness. Mistakes as to the severe forms of the condition are very easily made.

Conditions to be Differentiated from Acute Alcoholism

It may be mistaken for:
Uremia
Apoplexy
Concussion of the brain
Fracture of the skull
Poisoning by opium
Diabetic coma
Postepileptic coma
Narcotic poisoning.

UREMIA.

From uremia, the diagnosis is easily made by catheterization and examination of the urine; by blood pressure—which is high in uremia as a rule; and again, by the degree of unconsciousness—in alcoholism this is rarely as deep as in uremia. Respiration is apt to be slow in alcoholism, and frequently is of the Cheyne-Stokes type in uremia. The phenolsulphonephthalein test will show very little function of the kidney in uremia; it is likely to be much better in alcoholic coma.

APOPLEXY.

In apoplexy there is practically always a hemiplegia or other paralysis. The spinal fluid is often bloody and contains polymorphonuclear leukocytes.

CONCUSSION OF THE BRAIN—FRACTURE OF THE SKULL.

In these conditions there may be the history of injury. A characteristic symptom is a tendency, as the patient recovers, to turn from one side to the other, and to draw his knees up toward his chest. A fracture of the brain may be demonstrated or a paralysis exist. Examination of the eye ground will show choked disk in certain cases, with hemorrhage. It must be remembered that an alcoholic can receive a fracture of the skull. If there is reason for doubt, the case should be treated as one of fracture.

POISON BY OPIUM.

Opium poisoning can be distinguished by the pin-point pupils; very slow respiration, usually rapid heart and the history of having taken the narcotic.

DIABETIC COMA.

Diabetic coma is characterized by a deep, rather rapid inspiration—a veritable air hunger. Diacetic acid or acetone is found in the urine, though in cases of true diabetic coma the sugar may occasionally be overlooked.

POSTEPILEPTIC COMA.

This condition can be distinguished by marks on the tongue where the latter has been bitten, also by the history of epileptic seizures during the life of the individual.

NARCOTIC POISONING.

In belladonna poisoning there is often wild delirium, widely dilated pupils, flushed skin and dry mouth.

Alcoholic odor on the breath is not always to be depended upon, for the reason that a person may have been drinking alcohol, and then been overcome by one or the other of the accidents above described.

(*b*) *Chronic Alcoholism*

Symptoms.—Chronic alcoholism has as its result, symptoms referable to the muscular, nervous, renal, hepatic, cardiac and digestive systems.

Nervous System.—Almost any disturbance of the nervous system may be looked for—unsteadiness in gait, tremor, particularly of the hands and tongue, and dull mental processes. Peripheral neuritis is very common, as are hysterical attacks, particularly in women when coming out of an attack of alcoholism.

Of the *digestive symptoms*, gastritis, vomiting in the mornings, a feeling of distress in the morning until after a drink is taken, and dilatation of the stomach are common.

The hepatic system is affected particularly by alcohol acting as a tissue poison upon the liver cells, bringing about a destruction of the liver cells, a fatty degeneration, and an increase in the connective tissue—in other words there is a marked sclerosis preceding the actual symptoms and signs of sclerosis. The liver is apt to be large and tender, and often attacks of jaundice occur which are catarrhal in character.

Arteries.—Unquestionably alcohol is a causative influence in bringing about arteriosclerosis, although the studies of Cabot would possibly throw some doubt upon this question.

Conditions to be Differentiated from Chronic Alcoholism

This condition must be differentiated from:

Degeneration due to other poisons than alcohol
Tabes dorsalis
Senile tremor or tremor of paralysis agitans
Paralysis of the insane.

DEGENERATION DUE TO OTHER POISONS THAN ALCOHOL.

Degeneration due to other poisons than alcohol can be differentiated by discovery of the other drug. Hysterical attacks in women are extremely common after a bout or after excessive drinking, or as a result of an attempt to break off the habit, and are very deceptive. Frequently a woman attempts to hide the fact that she has been drinking; without this knowledge the physician constantly looks upon the case as one of simple hysteria. The history is all important. The fact that there is no patent reason for mental disturbance should be given full weight.

TABES DORSALIS.

This condition may be mistaken for alcoholic neuritis, but the presence of eye symptoms, the presence of a Wassermann reaction in the spinal fluid, together with an increase of the cells, will make the diagnosis positive for tabes.

SENILE TREMOR OR TREMOR OF PARALYSIS AGITANS.

This condition might be mistaken for alcoholism, but the advanced age of the patient in the first condition, the curious masklike expression of the face in the second, the absence of the drink habit, will mark the case as due to other causes than alcohol.

GENERAL PARESIS OF THE INSANE.

Paralysis of the insane may be simulated. Paresis is chronic in its course, is syphilitic in origin, there is defective memory for well-known facts, there are delusions of grandeur, there are speech disturbances; these are progressive in character.

Delirium tremens

This is the result of chronic alcoholism which is characterized by a maniacal condition which follows overdrinking in an alcoholic, or the sudden withdrawal of alcohol; it may be the result of a surgical shock, either the result of an operation or an accident in an alcoholic. The patient is restless, has visions which are usually unpleasant, talks constantly, and entirely incoherently; he imagines his friends are enemies, sees objects flying and crawling about, and often screams with fear. These patients always have insomnia, rapid pulse and dilated heart; extreme prostration occurs, and death often takes place from heart failure.

The curious terrors that the individual experiences, combined with the history of alcoholic habit—the tremor, the depression, and the dilatation of the heart—all these symptoms characterize this form of mania as a distinct type.

Diagnostic Factors.—It can be confused with the other forms of mania, but the short history of these, the absence of tremor and terrifying hallucinations all help to distinguish them from the alcoholic condition.

2. Opium Poisoning

Origin.—The opium habit is the result of constant use of small doses of opium or its derivatives. Soon the regular dose of opium becomes an apparent necessity to the individual, so that its use cannot be dispensed with without great difficulty. The origin of the constant use of opium in those who do not belong to the criminal class is usually in the administration of the drug, either by a physician, or by the patient himself for the relief of pain. Frequently the desire for the drug becomes so great that it is taken by the patient when no pain exists.

Influence of the Drug.—When under the influence of a *moderate dose* of the drug, the patient is alert, his eyes are bright, he thinks well, he is entertaining. A *larger dose* causes him to be somnolent, with contraction of the pupils and inability to think. *When the drug has been taken for a considerable time*, the skin becomes sallow, often tremors occur, sometimes the imagination becomes disturbed. The individual becomes entirely unreliable, starts to be untruthful, keeps bad company, and soon deteriorates into a worthless person. Often the patient does not sleep well, yet he is stupid and somnolent; sometimes there is contraction and at other times dilatation of the pupils; pains neuralgic in character are common, hyperesthesias occur; indigestion is common, and fetor of the breath is marked. If the patient uses the drug hypodermically, the body is often covered with marks of the needle.

When the patient is not under the influence of the drug, he is depressed, restless, taciturn and often has attacks of diarrhea and vomiting. The habit of opium-taking is particularly difficult to detect when the individual attempts to withhold the history of his habit, and notoriously the history is voluntarily frequently withheld by the patient. The drug is most frequently taken in the form of morphin, heroin, or codein, though it is often in the form of laudanum or paregoric, and frequently it is smoked. Of these, smoking appears to be the least severe form of poisoning, and the hypodermic use of one of the alkaloids the most severe.

Conditions to be Differentiated from Opium Poisoning

It may be mistaken for:

Hysteria

Various functional disturbances.

The diagnosis can only be made by the discovery of the habit.

HYSTERIA.

When a patient who is subject to the use of the drug for any reason is deprived of it, his disposition almost always changes. He then becomes extremely irritable, quarrels with anyone with whom he comes in contact, and may indeed have actual hallucinations. He resorts to all sorts of vague complaints, none of which can certainly be verified. Under these conditions the physician may well conclude the case is one of some functional disturbance, unless he is informed of or suspects the habit.

VARIOUS FUNCTIONAL DISTURBANCES.

Organic nerve conditions such as paresis and organic disease of the cord—especially locomotor ataxia—may be suspected, but they can be diagnosed by the fact that the real condition has certain and fixed symptoms, while the symptoms of morphin poisoning are most changeable. When a patient who is a morphin habitué is found unconscious, a careful differentiation must be made between that and other states of unconsciousness. Usually the best distinction is in the peculiarly slow respirations and contracted pupils of opium poisoning, but it must be remembered that the pupils may dilate in the last stages.

3. Lead Poisoning

Forms.—Lead poisoning occurs in acute and chronic forms.

(*a*) *Acute Lead Poisoning*

Acute lead poisoning has all the marks of any other irritating poison which has its first effects on the gastro-intestinal tract—vomiting, diarrhea, abdominal pain and collapse. For further information on this form of lead poisoning the reader is referred to the literature on toxicology.

(*b*) *Chronic Lead Poisoning*

Chronic lead poisoning, on the other hand, is usually quite insidious in its onset. Frequently there are abdominal pains—so-called lead colic—coming on in paroxysms, the pain being extreme in character and accompanied by obstinate constipation; diarrhea, instead of constipation, occurs very rarely.

Causes.—The cause of chronic lead poisoning is absorption of the lead by lead workers or by such artisans as painters. It is particularly to be remembered that laborers in iron mills complaining of abdominal pain are apt to be affected with lead poisoning, because they either handle iron which is freshly painted with red lead or they themselves paint the iron. When such laborers are asked if they handle lead they invariably deny the fact, but often the red paint can be seen about their hands and clothes. Makers of certain fabrics such as silks, and laces, in which lead is used in the manufacture of the material, may be affected. Water, not containing lime salts and being conveyed through lead pipes, and the adulteration of foods are fertile sources of poisoning. In Stewart's cases it occurred from the lead chromate used in cakes. The use of cosmetics has been known to cause lead poisoning.

Characteristic Features.—The chronic form of lead poisoning is apt to be accompanied by paralysis of any or all muscles, due to peripheral neuritis. It is particularly characterized by paralysis of the extensors of the arms, causing a double wrist drop. Occasionally there is very rapid anemia. The patient is yellow and cachectic in appearance. Examination of the blood shows a reduction of hemoglobin, a reduction of the corpuscles, and a marked basophilic degeneration of the red cells. A blue line on the gums is quite characteristic. It is due to a deposit of the sulphid of lead in the tissues of the gums, and is seen on the free margin.

Not only does the palsy affect the extensors of the arms, causing double wrist drop, but occasionally there is a single wrist drop, only the nerves of one arm being affected. Sometimes the legs are involved and foot drop occurs. Occasionally the paralysis is of the brachial type, when the deltoid, biceps and brachialis anticus muscles are affected—Duchene-Erb type. Sometimes it is the Aran-Duchene type, where the small muscles of the hand are involved and become atrophied; at times there is paralysis of the cranial nerves. Palsy may be general, often beginning as a wrist drop and ending as general multiple neuritis. Before an actual paralysis occurs the individual suffers moderate pain in various parts of the body, probably due to minute hemorrhages in the substance of the nerves. In the usual case the faculties are not all affected, but in certain fulminating cases the cerebral symptoms may be very marked; delirium occurs which may become maniacal in character. Coma may occur as the first symptom; hysterical symptoms are common and occasionally convulsions occur.

Tremor is extremely common in all the forms of chronic lead poisoning.

Lead is one of the acknowledged factors causing arteriosclerosis, high blood pressure, and interstitial nephritis.

The diagnostic signs are, abdominal pain, peripheral neuritis, painless in character, characterized by double wrist drop, a blue line upon the gums, and basophilic degeneration of the red cells.

Conditions to be Differentiated from Lead Colic

Lead colic may be mistaken for:

Acute perforating peritonitis

Acute local peritonitis (not perforating in type)

Gall-bladder disease

Renal colic (or indeed for almost any painful condition of the abdomen).

Abdominal Pain.—No case of abdominal pain should be considered completely diagnosed until a history is taken, with the especial idea of discovering the possibility of lead absorption, and until examination of the gums is carefully made, to positively establish the fact that no lead line is present. More than one case has been saved from operation by *examination of the gums* which show a blue line of lead, and by a blood examination showing degeneration of the red cells. In cases of abdominal pain due to inflammatory conditions there is fever, prostration and leukocytosis, none of which occur in lead poisoning.

Neuritis.—The peripheral neuritis may be mistaken for any peripheral neuritis due to any other toxic substance.

When the wrist drop is seen, it is very likely to be mistaken for a pressure palsy, but pressure palsy is unilateral, while lead palsy is usually bilateral. Examination of the gums and of the blood will put the physician upon the proper track for diagnosis.

Alcoholism is a very common cause of chronic multiple neuritis, and at the same time is a predisposing cause for lead absorption. It is therefore of the highest importance to distinguish between *alcoholic peripheral neuritis* and *neuritis due to lead with alcoholism as a simple causative factor.* Here, as repeatedly stated, the finding of a lead line, of basophilic degeneration of red cells, and in rare cases the presence of lead in the urine will make the diagnosis.

NERVOUS SYMPTOMS AND MANIA.—The nervous symptoms and the mania may be mistaken for acute mania, general paresis, epilepsy, meningitis and uremia, but these conditions can be excluded by the absence of blue line on the gums, the blood signs, the history of the case, and finally, in doubtful cases, by the examination of urine for lead.

4. Arsenical Poisoning

Symptoms.—Arsenical poisoning is characterized in the acute form by symptoms of acute gastro-enteritis, vomiting, abdominal pain, diarrhea, and great prostration. The diagnosis of any other gastro-enteric inflammation and that caused by arsenic must depend, firstly, upon the history of the drug that has been taken—if such history is obtainable; if not, it must depend upon an examination of the patient, and finally upon examination of the urine which will show the presence of arsenic by Marsh's test, when the gastro-enteritis is due to arsenic.

Chronic Arsenical Poisoning.—Chronic arsenical poisoning may occur in the arts; it may be the result of taking medicine or the use of arsenic as habit, or it may be the result of arsenic absorbed from wall papers and other unusual sources.

The symptoms of those *patients working in the arts* who are affected by arsenic are practically those of patients suffering from neuritis. The peripheral neuritis may be so severe that there is tingling in the hands and feet; there is loss of the patellar reflexes and interference with locomotion.

In the *cases caused by the drug used as a medicine,* the first symptoms of poisoning are puffiness of the face, colicky pains in the abdomen, and a tendency to diarrhea. If these symptoms do not appear, or if they are disregarded, then pigmentation of the skin and a *multiple neuritis* are apt to follow. One of the author's cases, a patient suffering from a secondary anemia, was given as large a dose as 30 drops of Fowler's solution three times a day; she developed both skin lesions and multiple neuritis.

Conditions to be Differentiated from these Cases of Multiple Neuritis

These cases of multiple neuritis must be separated from those due to other conditions such as:

Alcoholic neuritis
Lead poisoning
Beriberi.

No case so arising should be considered complete in the diagnosis, unless the history is carefully taken.

ALCOHOLIC NEURITIS.

Alcoholic neuritis, almost without exception, gives a history of alcohol. As a rule there is but little pain, and the appearance of the patient is that of an individual who is an habitual user of alcohol.

LEAD POISONING.

Lead poisoning can be differentiated by a blue line upon the gums, and a stippling of the red cells.

BERIBERI.

Beriberi has edema or emaciation as part of the symptomatology. It usually occurs in regions where polished rice is used as food. There may be a history of early infectious symptoms.

5. Food Poisoning

An article which is, as a rule, perfectly wholesome food may become poisonous when it contains chemical or bacteriological material which affects the organism in a deleterious manner. Certain articles of food may be wholesome when first prepared and become poisonous, due to changes occurring after preparation. Certain mushrooms and fish are always poisonous. Certain fish and meat are poisonous when they contain pathogenic organisms or bacteria. Fish and meat containing ova of tapeworm, milk containing germs of typhoid fever and tuberculosis, cheese and milk undergoing chemical and bacteriological change, may be cited as well-known instances of usually wholesome food becoming poisonous.

FISH.—Certain fish, especially those found in eastern waters, are always poisonous. The fungi poisons resemble curare in its effects. Canned fish becomes poisonous occasionally because it undergoes bacteriological change and not because it absorbs metallic substances from the container. In these days of remarkable advance in preservation of food, poisoning by lead and tin is of rare occurrence; indeed, all canned goods prepared by reliable firms are safe articles of food.

Oysters have frequently been the source of starting and spreading epidemics of typhoid fever. There are other shellfish that are in themselves poisonous, but they are usually so because they are undergoing some bacteriological change.

Meat.—According to Novy, there are two forms of meat poisoning which are sharply contrasted. In the first the central nervous system is affected and the symptoms therefore are characteristic and well marked. Owing to the frequency of these symptoms among the recorded cases of sausage poisoning, van Ermengem designated this condition as a true botulism. In the second form of intoxications the symptoms are gastro-intestinal. They may be of a mild type and of short duration, or of a more severe type, merging into a severe infection.

Vegetables.—Certain vegetables give rise to poisons, such as ergot and mushrooms.

Symptoms.—The symptoms of these various poisons are varied. For instance in fish poisoning due to the toxins caused by bacteria there is general weakness, dull pain in the abdomen, dyspnea, mydriasis, diplopia and vertigo, complete dryness of the mouth and tongue, inability to swallow and loss of speech. Vomiting and diarrhea are absent, and instead there is obstinate constipation and retention of urine. On the other hand certain cases are of the type of an acute gastritis, with vomiting, diarrhea, dizziness, tremor, prostration and cardiac syncope. These cases are *actual infections;* the others are due to *toxic poisons.*

Diagnosis.—The point in diagnosis in these various cases must be the history of the case, and therefore when a case of acute enteritis or a case evidently due to some toxin occurs, particularly in epidemic form, it is extremely important to attempt to trace this source of poisoning, not only to make a diagnosis, but to prevent a repetition of the occurrence. Of these, poisoning by foods must be differentiated from poisoning by various other substances.

Poisoning by Arsenic, for instance, gives rise to acute gastritis, with all of the symptoms which occur in the form of poisoning from food due to an actual local infection of the parts affected. Here, however, arsenic can be obtained both from the food and from the urine. Poison from tartar emetic gives rise to acute gastro-enteritis.

Poison by Atropin gives rise to symptoms not unlike the symptoms due to toxins originating in various foods.

These sources of poisoning must be excluded before the symptoms can be actually attributed to the food.

6. Pellagra

Cause.—This is a disease the cause of which is unknown.

Occurrence.—*Age.*—It occurs most commonly in persons from twenty to fifty years of age.

Sex.—In America there are many more females than males affected.

Season.—It appears in the spring and autumn.

General Symptoms.—It is characterized by gastro-intestinal disturbances, by skin lesions and by changes in the nervous system.

Etiology.—It is frequently epidemic. The disease is connected with bad food and bad water, and severe labor. According to Shaw, it is unquestionably connected with the ingestion of bad maize. The exact cause as stated above is not known.

The most severe effects of the poison fall upon the nervous system. Erythema and the gangrene are believed to be secondary to these nervous changes. The skin lesions are necessary for a diagnosis.

Special Symptoms.—The symptoms may be conveniently grouped into those of the skin, the alimentary canal and the nervous system, though they do not follow any sequence, according to Beall. One case for a long while may resemble simple neurasthenia and finally develop gastro-intestinal or skin symptoms, or skin symptoms resembling sunburn may appear at several recurring spring seasons, or a long-standing stomatitis may be the first symptom. Wood believes no case can be diagnosed without the presence of skin manifestations.

Skin.—First as to the skin. There is a symmetrical and erythematous eruption which occurs on the parts exposed to the sun. The skin is swollen, burning and itching; petechiae are frequent, and bullae appear. About two weeks after the beginning of the erythema, it subsides and desquamation follows. Usually these skin eruptions disappear in the autumn and reappear in the spring.

Gastro-intestinal Symptoms.—The gastro-intestinal symptoms consist in stomatitis and diarrhea with other gastro-intestinal symptoms of indigestion.

Nervous Symptoms.—The nervous symptoms consist first in a melancholia—the delirium may be maniacal in character; there may be twitchings, tremors or epileptic seizures. Frequently the patellar reflex is increased. The gait is not ataxic, but more like that of spastic paraplegia. Ankle clonus is often present.

These paretic symptoms are commonly preceded by tremor. Sensation is not affected. The patient loses strength and flesh. As years go on his skin shrivels and the bones become prominent; the muscles waste and the movements are slow and languid. The disease at times is quite chronic; it is rarely very acute.

Conditions to be Differentiated from Pellagra

This condition can be mistaken for:

Psychoses
Sunburn

Leprosy
Chronic gastro-enteritis
Addison's disease
Syphilis.

PSYCHOSES.

The various psychoses can only be differentiated by the absence of other signs of pellagra. Without the history of a symmetrical skin lesion or an intermittent but chronic gastro-intestinal condition, the psychoses are probably independent in origin, but combined with the presence of a typical skin lesion, the diagnosis points toward pellagra.

SUNBURN.

Many cases of pellagra first show the skin lesion on exposure to the rays of the sun. But sunburn is not necessarily bilateral and symmetrical and disappears after proper treatment and protection from the sun; neither is it accompanied or followed by gastro-intestinal or mental disturbances.

LEPROSY.

The history of these two conditions—leprosy and pellagra—is entirely unlike. An examination of a piece of excised skin in a case of the former will show the bacilli of leprosy.

CHRONIC GASTRO-ENTERITIS.

Chronic gastro-enteritis may be present in true cases of pellagra for a considerable time before the pathognomic skin lesions or the psychosis occurs. No distinctive sign except the absence of either psychosis or skin lesions will enable one to make a diagnosis of gastro-enteritis independent of pellagra.

ADDISON'S DISEASE.

Pellagra might be mistaken for Addison's disease, but this is a mere pigmentation and there is no stomatitis; there is also the presence of tuberculosis in other parts of the body. The curious lack of ambition, the presence of weakness which is so characteristic of Addison's disease, and the very rapid heart of Addison's disease help to distinguish it from pellagra.

SYPHILIS.

The characteristic features of syphilis are a history of a primary sore, a Wassermann reaction, and the fact that the patient recovers under antisyphilitic treatment.

7. Beriberi

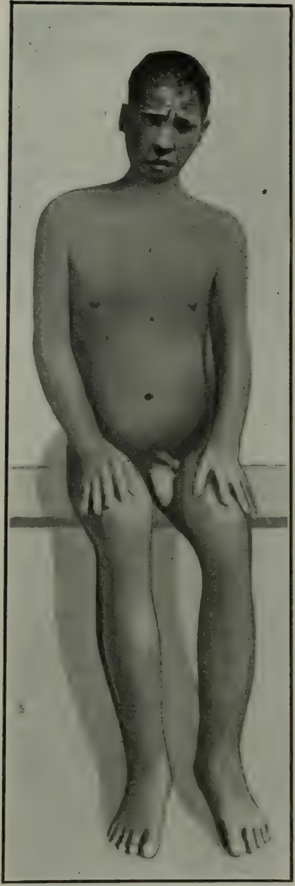

Fig. 45.—Beriberi, Showing Edema. (From Modern Medicine, Vol. II.)

Cause.—There are two views as to the cause of this disease, one being that it is an infection, the other, that it is a disease of metabolism due to faulty food intake. Whatever the actual cause of the disease, unquestionably it has to do with eating of certain kinds of rice. Persons who eat polished rice are the individuals who are affected. In polished rice there is absence of vitamins from the food. People who are fed upon unpolished rice or upon parboiled rice never develop the disease; in individuals who are developing the disease, when put upon a diet of unpolished rice or when given parboiled rice, the disease entirely disappears, and the entire epidemic ceases to exist.

Symptoms.—Beriberi is characterized in its *early stages* by the ordinary symptoms of an infection—vomiting, depression and slight fever. In its *terminal stages* it is characterized by a general multiple neuritis, sometimes with edema, great exhaustion and, frequently, by death.

Forms.—Authors have described two forms of the disease, (1) the so-called *wet form* in which there is much edema (Fig. 45), and (2) the *emaciated form* in which all the muscles become atrophied and the individual becomes entirely helpless and dies (Fig. 46).

Diagnosis.—The diagnosis of the case will depend upon the history or presence of multiple neuritis, of edema or emaciation in those individuals who have partaken of polished rice. It

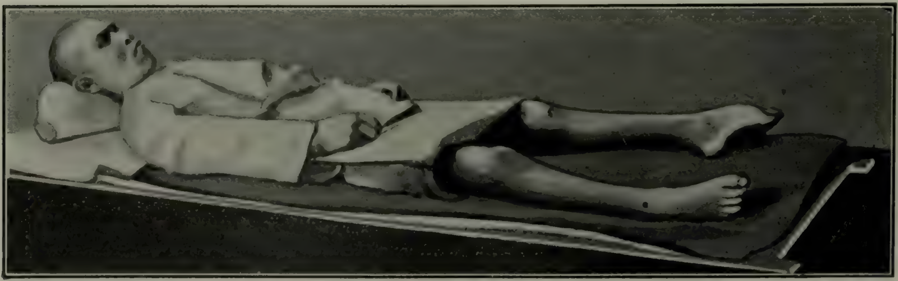

Fig. 46.—Beriberi, Showing Muscular Atrophy. (From Modern Medicine, Vol. II.)

is perhaps entirely impossible to diagnose the disease in the very early stages except in the presence of an epidemic.

Conditions to be Differentiated from Beriberi

Beriberi might be mistaken for the following conditions. Some of the special diagnostic symptoms are given below:

Multiple alcoholic neuritis
Arsenical multiple neuritis
Poliomyelitis
Landray's paralysis
Anesthetic leprosy
Tabes dorsalis.

Multiple Alcoholic Neuritis.

In multiple alcoholic neuritis there is no edema, and the other characteristics of beriberi are entirely wanting. There is the history of alcoholism.

Arsenical Multiple Neuritis.

Arsenical multiple neuritis might also be mistaken for this condition, but there is no history of the antecedent infectious stage.

Poliomyelitis.

Certain forms of beriberi resemble rather closely the *bulbar form of epidemic poliomyelitis,* though this condition can scarcely be mistaken for any other. The paralysis which occurs in epidemic poliomyelitis is central and not peripheral in origin. It is very frequently monoplegic; the neuritis of beriberi is general.

Landray's Paralysis.

In Landray's paralysis there is fever and acute symptoms at first; there is no disturbance of sensation. In these particulars, it differs from beriberi; it is also much more acute in its course.

Anesthetic Leprosy.

Anesthetic leprosy shows thickened skin, ulcerated areas, and true anesthesia—not hyperesthesia, as is likely to be the case in beriberi.

In none of the above conditions is the history of eating polished rice to be found.

Section IV

Diseases of Metabolism

1. Gout

(*Podagra*)

Definition.—Gout is a disease due to disturbed metabolism, in which the disturbing element is the retention of one or all of the purin bodies, among which is uric acid. It is characterized mainly by attacks of acute arthritis.

Etiology.—There is still much divergence of opinion as to the true cause of this disturbance of metabolism. Certain cases, however, are distinctly hereditary, others appear to have overeating—particularly of meat—and overindulgence in alcohol—especially of malt liquors—as their cause.

Occurrence.—More males are affected than females. The disease is common in England and Germany, but not at all rare in the United States. Many cases occur in every doctor's general practice.

It is not limited to the rich: it is also common among the moderately well-to-do and among the actually poor. According to Osler, "poor man's gout" is a common affection caused by neglect, underfeeding and excessive consumption of malt liquors.

Classification.—The attacks of gout may be conveniently divided into: acute typical gout, chronic gout, and atypical or irregular attacks of gout.

ACUTE TYPICAL GOUT.—A typical attack of gout is characterized by certain prodromal symptoms such as indigestion, headache, malaise and twinges of pain. The actual attack, almost without exception, begins suddenly during the night. There is sharp fever, ranging between 100° and 103° F.; the metatarsal phalangeal joint of the great toe, usually on the right side, becomes suddenly swollen, red, edematous and extremely tender. During the following day there is an amelioration of all of the symptoms: the fever drops and the joint becomes less tender and painful, although it is still swollen and tender and the surrounding tissue edematous.

The following night all the symptoms recur, to again improve the next day. This second attack is not likely to be quite so severe as the first, and so, during a period of from three to seven days, the attacks recur with gradual tendency toward recovery.

As the joint becomes less painful the swelling decreases, the glistening appearance of the skin disappears, as does the edema, and the integument becomes wrinkled, finally undergoing considerable desquamation at the end of the attack. During the attack the patient is querulous and ill-tempered; the appetite is usually retained.

The attack over, the patient is seemingly as well as ever until other seizures follow at intervals of days, weeks or even years. With repeated attacks, however, there comes about some anatomical change in the joints affected, due to the deposit of biurate of soda in and about the joint. Other joints than the toes are affected—the fingers are particularly prone to the affection; around them also there is the same deposit of biurate of soda.

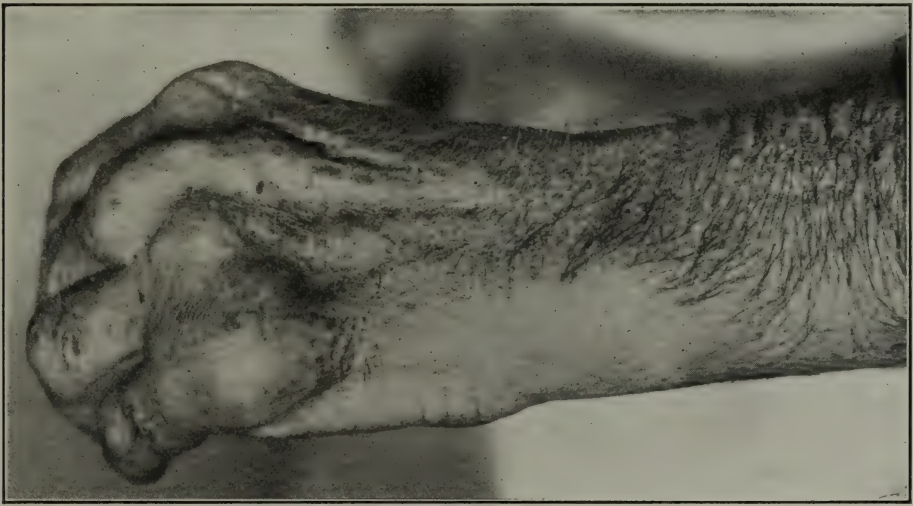

Fig. 47.—Tophi of Gout—About Metacarpophalangeal Joints. (Personal Observation.)

Frequently there are tophi or chalklike deposits of the biurate in the various cartilages, the outer edge of the pinna of the ear being a favorite spot. These deposits are hard, calcerous masses which give rise to no symptoms in such places as the ear, but when they occur in or around the joints and interfere with their movements they are the cause of considerable trouble. They are of the greatest diagnostic value. Occasionally during the attacks of acute typical gout there is a rather sudden subsidence of the joint symptoms with vertigo, dyspnea, pains in the region of the heart and even apoplectic symptoms—the so-called *retrocedent gout.* When the disease has lasted a long while, sclerotic changes take place in the kidney, the heart, and the blood vessels.

Chronic Gout.

Chronic gout is characterized by arthritis affecting the smaller joints, usually though it may affect any of the joints. The arthritis is peculiar in that about the joints is a true calcerous exudate made up of the biurate of soda. (Fig. 47.) Often the exudates are quite superficial and may ulcerate through the skin. (Fig. 48.) These same urate deposits may take place in almost any cartilaginous tissue or connective tissue of the body, and they may also be found in the skin.

Sclerotic changes occur in the various organs causing interference with their functions. These changes are not to be distinguished from sclerotic changes due to other causes, except by the history of the case as having been one of true gout. These sclerotic and vessel changes bring about a train of symptoms that are caused by other reasons than gout.

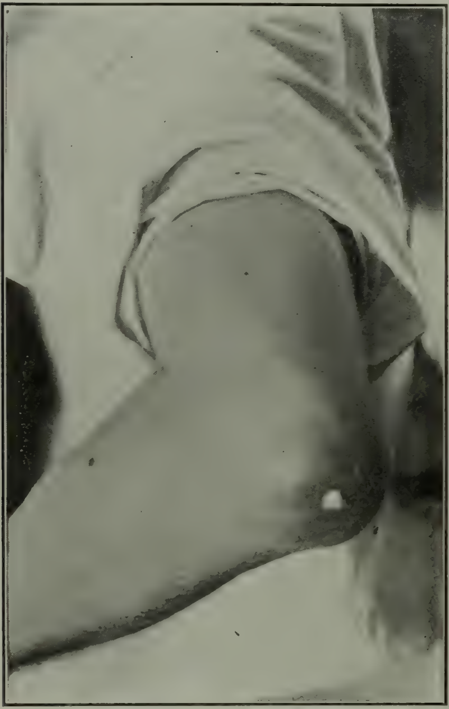

Fig. 48.—Broken-down Typhus at Elbow Joint. (Personal Observation.)

Atypical or Irregular Gout.

Atypical or irregular gout consists in eczematous outbreaks, in various neuralgias and headaches and attacks of inflammation of the eye —particularly in the sclera and conjunctivae.

Conditions to be Differentiated from Gout

The disease is to be distinguished from the following conditions:
Arthritis due to traumatism
Rheumatism
Arthritis deformans
Arthritis due to grave nerve changes (arthropathies)
Pulmonary arthropathies
Arthritis due to infectious conditions other than rheumatism.

Arthritis Due to Traumatism.

The history of a traumatism is always present when that is the cause; the traumatism causing arthritis may affect any joint. In gout there is no history of traumatism, but there is the history of repeated attacks of joint pains, particularly in the great toe; occasionally there is real difficulty in making this differentiation in the first attack, when the joint affected is that of the great toe, and the history of injury is incomplete.

Rheumatism.

Rheumatism frequently attacks the larger joints, gout the smaller, particularly the great toe, and the pain proceeds from one joint to another until all of them may be involved. There is much prostration and leukocytosis occurs; these are absent in gout.

A sudden attack of pain in the great toe, as described above, is very apt indeed to be *gout* and not rheumatism. In the latter there may be accompanying endocarditis; this does not occur in gout. Rheumatism frequently responds promptly to salicylates, which are not of so great value in gout. In gout, particularly after the first attack, tophi may be present on the joints or ears, while they are not present in rheumatism. A tonsillitis is frequently the first indication of an attack of rheumatism.

Arthritis deformans.

The greatest difficulty of differentiation is perhaps in rheumatoid arthritis; this disease may begin acutely, simulating an acute attack of rheumatism affecting the smaller joints. It does not as a rule attack the feet first and is extremely rare in the great toe alone.

In chronic cases of gout, after the hands and feet are both attacked, the diagnosis can be made upon the history of previous attacks of true gout affecting the great toe perhaps many years back, and other attacks having been repeated. Heberden's nodes which affect the interphalangeal joints are fibrinous and not tophi. There is no deposit of biurate of soda in or about the cartilages of rheumatoid arthritis.

2. Diabetes mellitus

Cause.—Diabetes mellitus is a disease due to a disturbed condition of metabolism, in which there is excessive polyuria, the urine continuously containing greater or lesser amounts of glucose (grape sugar).

Characteristic Symptoms.—The characteristics of this disease are polyuria, thirst, increased appetite, dry mouth and tongue, pruritus in various portions of the body—particularly about the genitals—furunculosis and emaciation. The urine, as stated above, is of high specific gravity, light green tinge, and continuously contains glucose.

When the disease affects the young it is extremely serious and frequently fatal. There is always great emaciation under such conditions. Older individuals may pass large quantities of urine containing large amounts of glucose, and except for the inconvenience caused by the polyuria and perhaps by the itching, the disease may last for years with no serious results. But no individual who has true diabetes mellitus is free from danger of some of the sudden serious and often fatal complications. *Cases may exist also for years without the knowledge of the person affected and without the possibility of the doctor in attendance making a diagnosis, unless he makes routine urine examinations of all the cases which come under his care.*

Complications.—Some of the serious complications which occur are gangrene of any portion of the body, acidosis, diabetic coma. A person in apparently perfect health may have a very slight injury, and the part become gangrenous, or the gangrene may occur without any known injury, usually affecting a toe or a finger. Examination of the urine will probably show the continued presence of glucose.

A case of diabetes known to be such, may be progressing apparently normally, when because of overwork, error in diet, mental strain, etc., the patient will have sudden attacks of headache, of vertigo, general weakness and somnolence. Examination of the urine under these conditions will often show the presence of glucose, diacetic acid and acetone. Or the case may suddenly become unconscious, with long drawn inspiration, with forcible expirations, the breathing rather hurried, the patient in a typical condition of "air hunger." Possibly at first he may be aroused, but will immediately fall asleep again when left alone. In these conditions also there is usually the presence of sugar, diacetic acid and acetone in the urine.

Conditions to be Differentiated from Diabetes mellitus

The disease must be distinguished from several conditions. There may be other substances than "grape sugar" in the urine which will reduce Fehling's solution, or there may be a lowered tolerance to carbohydrates without the individual being a true diabetic.

The diagnosis is made positive *by the passage of large amounts of urine, continually containing glucose, when the patient is upon a diet containing only a small amount of carbohydrates.*

Substances besides glucose which will reduce Fehling's solution are milk sugar, glycuronic acid compounds, pentose, levulose, maltose and some inorganic substances of normal urine, among which are creatinin, uric acid, allantoin, nucleo-albumin, pyrocatechin, hydrochinin, and bile pigment.

If salicylic acid, glycerin, benzoic acid or sulphonal, chloral, camphor, naphthol have been given to the patient the urine will reduce Fehling's solution.

Where the reducing substance is any one of the above, the specific symptoms which belong to diabetes mellitus are *not* present in these cases

—to wit: thirst, polyuria, furunculosis, etc., but the urine of all such patients will reduce Fehling's solution in a more or less characteristic way.

Such conditions can only be detected *by routine examination of the urine of every patient presenting himself.* When urine is found to reduce Fehling's solution, and no symptoms present themselves, means should at once be taken to be certain whether the reducing substance is glucose. The fermentation test should be employed; if glucose be present the urine will ferment upon the addition of yeast, driving the urine from the fermentation tube. The specific gravity of the urine will be reduced by the fermentation. If such fermentation does not take place, the reducing factor is one of the various substances named.

It is a fact that levulose and maltose will also ferment when the urine containing them is mixed with yeast and the mixture kept in a warm place for twelve hours. If the case has none of the signs or symptoms of diabetes and the urine ferments with yeast, the specific tests for glucose, levulose and maltose must be used. The reader is referred to special books on urinary diagnosis for the methods to be followed.

Pregnant women shortly before the birth of a child, or nursing women a few days after lactation begins, sometimes secrete *lactose,* which resembles glucose in its reducing power, but which *cannot be fermented by yeast.*

Malingering must also be considered. Patients have added grape sugar to the urine for the purpose of deception. Only a detection of the fraud and the absence of symptoms in the case which are common to diabetes, will prevent error.

ALIMENTARY GLYCOSURIA.

Occasionally persons taking largely of carbohydrates, secrete glucose in the urine. There may be symptoms at first which entirely disappear and do not return until another error in diet, or there may be no such symptoms. Such cases should *not* be looked upon as true diabetes mellitus, but should be considered as cases in which there is *some error in carbohydrate metabolism.*

One must here insist that while cases exist as above described, the physician must recognize that there is *a distinct inability in the individual affected to take care of carbohydrates ingested.* Such cases must frequently be carefully examined, because the appearance of sugar in a perfectly normal individual does not occur. The so-called alimentary glycosuria may be the first indication of a real diabetes mellitus.

DIABETES INSIPIDUS.

In these cases patients pass enormous amounts of urine of low specific gravity, free from albumin and tube casts, also free from sugar. Frequent micturition, backache and thirst are prominent symptoms. Careless observation or lack of all observation is responsible for mistaking these cases for diabetes mellitus.

3. Diabetic Coma

This condition is often the end result of diabetes mellitus and is preceded by a state common in other conditions than diabetes—to wit: acidosis.

ACIDOSIS is suggested by the occurrence of a sweet odor to the breath and by the presence of acetone bodies in the urine—these bodies are ACETONE, DIACETIC ACID and BETA-OXYBUTYRIC ACID.

Every case of diabetes mellitus under observation should *have the urine examined not only for sugar but for acetone and diacetic acid.* Diacetic acid and acetone will occur in the urine of diabetics, when they are first put upon a strict carbohydrate-free diet without their presence, indicating the danger of coma.

Diacetic acid can be diagnosticated by the addition of diluted solution of ferric chlorid to the urine, which will cause a brown discoloration. This same discoloration will be present in the urine of patients taking aspirin, salicylic acid and antipyrin, and this discoloration must not be mistaken for diacetic acid. Heating the urine before the addition of ferric chlorid will cause the diacetic acid reaction to disappear, while heating the urine of patients taking the drugs will have no effect on the reaction.

Acetone has a specific test in the use of nitroprussid of soda. The reader is referred to books on urinary analysis for the details.

The onset of the coma is further shown by the deep drawn inspiration; the patient becomes weak, quiet and drowsy, and falls into a coma in which he usually expires.

Conditions to be Differentiated from Diabetic Coma

The coma may be mistaken for the:

Coma of uremia
Apoplexy
Alcoholism
Opium poisoning
Head injury
Brain tumors
Meningitis.

None of these have the curious deep inspirations and expirations due to diabetic coma, and none of them have the acetone bodies and sugar in the urine.

In this manner they can be differentiated as well as by the symptoms and physical signs peculiar to each.

4. Diabetes insipidus

Characteristic Features.—Diabetes insipidus is a disease characterized by extreme polyuria, with urine of a specific gravity 1.000 to 1.004, containing no albumin and no casts and no glucose. This polyuria continues over a long space of time. There is a primary and a secondary form of diabetes insipidus; many of the cases are believed to have syphilis as the etiological factor. Besides the polyuria and low specific gravity, the patient complains of backache and extreme thirst.

Eye.—Often accompanying the condition there is a curious pigmentation of the choroid coat of the eye; never retinitis.

Heart.—The heart is normal in size and there is no accentuation of the second sound of the heart.

Urine.—The urine is pale, watery, of large quantity, with specific gravity 1.000 to 1.004; it rarely contains any abnormal ingredients.

General.—There is very little, if any, interference with the general health, unless the condition is the result of a cerebral tumor or the expression of a severe syphilitic condition.

Where there is an organic disease of the brain or cord, of necessity the urinary symptoms are merely secondary.

Conditions to be Differentiated from Diabetes insipidus

The disease must be distinguished from:

Polyuria following typhoid fever
Hysteria
Interstitial nephritis.

POLYURIA FOLLOWING TYPHOID FEVER.

In typhoid fever there is frequently extreme polyuria. This must not be confused with the disease diabetes insipidus. The symptoms of typhoid fever are present.

HYSTERIA.

Hysteria, or even great nervous excitement, is constantly followed by the passage of large amounts of urine resembling that of diabetes insipidus in every particular. Here there is the history of nervous excitement, or of long standing hysteria, and there are the ordinary stigmata of hysteria. The polyuria lasts only during the hysterical attack, the urine being normal in the intervals.

INTERSTITIAL NEPHRITIS.

Interstitial nephritis is recognized by the character of the urine. This is rarely as great in quantity as is the urine of diabetes insipidus, but may be as much as three or four quarts in twenty-four hours. Its specific gravity ranges from 1.004 to 1.012, therefore not nearly so low as in diabetes insipidus. Without exception it contains albumin, and tube casts may practically always be found if the urine is centrifuged.

There is hypertrophy of the left heart, also extremely high blood pressure, and the second aortic sound is accentuated. The patient is distinctly unwell—dyspnea, vertigo, and cardiac palpitation being the main symptoms. The absence of the Wassermann reaction will help to exclude diabetes insipidus.

5. Rickets

(*Rachitis*)

Cause.—Rickets is a condition in which there is a fault in metabolism, causing a deficiency of the lime salts and bone-forming elements of the body.

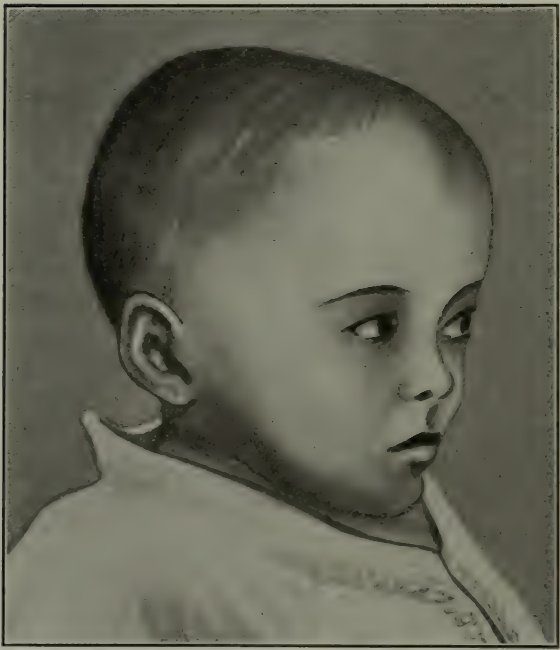

Fig. 49.—Head of Child with Rickets. (From Holt's Diseases of Children.)

Occurrence.—The disease occurs in infants, and always develops after birth.

Characteristic Features.—It is characterized by emaciation, irregular fever, enlargement of the ends of the bones at the junction of the diaphysis and epiphysis, particularly at the ankles, knees and wrists, and the external ends of the ribs. The fontanels close very slowly, the head enlarges, the forehead becoming straight and square, with parietal eminences. The bones are soft so that the effect of the pull of the muscles is to make the child bow-legged and bow-armed; there are continued sweats, particularly at night; the teeth appear late and decay early; there is a tendency to attacks of laryngismus stridulus, also attacks of tetany; there usually is enlargement of the liver and the spleen, with more or less severe anemia.

This condition is particularly common in artificially fed children, especially in those fed upon patented foods, sterilized milk, or excess of carbohydrate food; in other words upon food in which the elements of normal food are lacking or destroyed. It is extremely rare in breast-fed children. Defective hygiene also plays an important rôle in the development of rickets.

The child does not walk until late, often being twenty months or two years old (Fig. 49).

A typical "rickety" child has a large head with square forehead, bowed legs and arms, markedly prominent belly, enlarged spleen, pigeon-shaped chest; the fontanels are not closed until late; it is characterized by occasional attacks of fever.

Conditions to be Differentiated from Rickets

The condition is to be distinguished from:

Scurvy
Tuberculosis
Mere wasting due to improper food
Achondroplasia
Infantile paralysis
Hereditary syphilis.

SCURVY.

In scurvy there is marked enlargement of the shafts of the bones which are extremely painful and tender, in marked contrast to the painless and large joints, bowed legs and arms and beaded ribs of rickets. X-ray will show the enlargements along the shafts of the bones characteristic of scurvy, which are not present in rickets. This is acute in onset and follows the taking of improper food. Deprivation of fresh foods or the use of artificial foods in children are potent as causes of scurvy.

TUBERCULOSIS.

In tuberculosis there is marked emaciation without enlargement of spleen, there is lacking the bowing of the legs, the head is not enlarged or square, there is no "beading" at the ends of the ribs, there is no enlargement of the ends of the joints. Localization of the tuberculous process may be found in the lungs or the large mesentery glands, the latter causing tabes mesenterica. The kyphosis in rickets is often great and may resemble caries of the spine. Caries of the spine, however, is characterized by fixation of the spine, much tenderness over the prominence, with great amount of pain. These are absent in the kyphosis of rickets.

MERE WASTING DUE TO IMPROPER FOOD.

Simple marasmus, or atrophy due to improper food, is characterized by marked emaciation, by attacks of gastro-intestinal disturbances, and by the absence of enlarged spleen, but lacks the head and joint symptoms of rickets. The history of underfeeding can be obtained.

ACHONDROPLASIA.

This is an inherited intra-uterine disease. It is characterized also by bowed legs and arms and enlargement of the ends of the joints, but it is not a fault of nutrition. The enlargement is in the bone itself and not in the cartilaginous parts as in rickets. Indeed there is no cartilage formed. The child walks late and is one of the class of dwarfs which are so common; the arms are short; the individual is pug-nosed, the cranium is of normal size (Fig. 50).

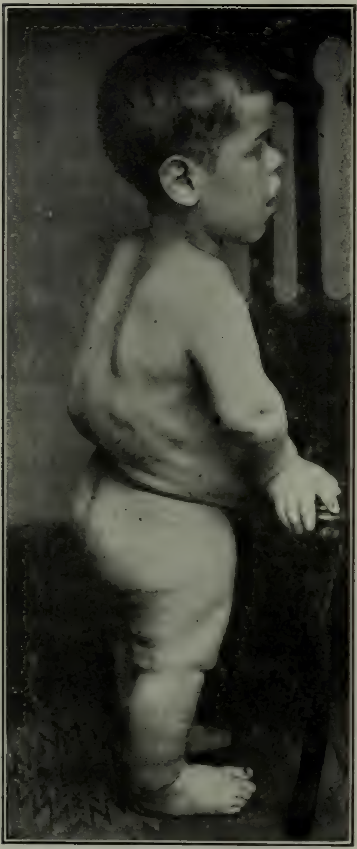

Fig. 50.—Achondroplasia. (Personal Observation.)

INFANTILE PARALYSIS.

Infantile paralysis, by its deformed limb in late stages, might be mistaken for rickets, but here the limb or limbs are paralyzed and atrophied. The limbs of a rachitic individual are not atrophied.

6. Scurvy—Scorbutus

Cause.—Scurvy is a condition caused by the *absence of certain substances in the diet.* This substance, *vitamin,* is present in fresh food, fresh meats and vegetable juices; hence the disease does not occur when these foods are used.

Occurrence.—It appears in both infants and adults. In infants it is apparently mistaken for other conditions, simply because the physician in attendance forgets that *children may develop scurvy.* Its cause in infants is deprivation of the mother's milk, and the use of food which does not contain the proper elements. The most deleterious foods are patented foods, sterilized milk and condensed milk.

Characteristic Features.—*In adults* the disease begins insidiously. The patient appears ill, is drowsy, short of breath and anemic. The characteristic changes—and those almost always present in the mouth—are particularly, softening and bleeding of the gums, ulcerations about the margins of teeth and necrosis of the alveoli. Soon hemorrhages occur, particularly under the skin and from the mucous membranes; the patient becomes extremely anemic, with a form of secondary anemia.

In children the disease is always characterized by fretfulness; there is pain and swelling of the legs, particularly over the tibia, due to subperiosteal hemorrhages. There is fever; sometimes there is swelling of the gums. Fretfulness and fever are found to be largely due to hemorrhages under the periosteum. Change of food and fruit juices will bring about immediate cure.

Conditions to be Differentiated from Scurvy in Children

Articular rheumatism
Periostitis
Infantile paralysis
Anemia from other causes
Ulcerative stomatitis.

ARTICULAR RHEUMATISM.

Rheumatism in children is characterized by swelling of the articulation; there is no hemorrhage. The kind of food has no effect upon the disease, and a change of food in a child with rheumatism does not bring about any change in the condition. If the condition be scurvy, however, and the food is properly regulated, the child becomes well in a short time.

PERIOSTITIS.

Periostitis may give the same local symptoms as scurvy, but in the latter swelling of the limbs is almost always bilateral, which is not the fact in periostitis. There is some fever, some leukocytosis; there is no history of improper food. Periostitis is not affected by change of food.

INFANTILE PARALYSIS.

Infantile paralysis in its early stages gives rise to great pain upon movement. This can be differentiated from scurvy by the fact that the loss of motion is a true paralysis and develops early; there is no error in diet; there is a great amount of leukocytosis, and there is little anemia.

ANEMIA FROM OTHER CAUSES.

Anemia from other causes is more apt to have subcutaneous hemorrhages, there is more apt to be bleeding from the kidneys and from the mucous membranes. The cause is not improper food.

ULCERATIVE STOMATITIS.

Ulcerative stomatitis affects the entire mucous membrane of the mouth; there is not the same bleeding of the gums; neither is there the dietetic error as in scurvy.

Conditions to be Differentiated from Scurvy in Adults

Purpura hemorrhagica
Mercurial stomatitis
Acute lymphatic leukemia.

PURPURA HEMORRHAGICA.

From purpura hemorrhagica the disease is differentiated by the fact that a purpuric individual has not been exposed to errors of diet or hardship; also by the fact that hemorrhages do not take on inflammatory conditions.

MERCURIAL STOMATITIS.

In mercurial stomatitis there is a history of having taken mercury or having worked with it. There is some salivation; there are not the characteristic hemorrhages.

ACUTE LYMPHATIC LEUKEMIA.

Acute lymphatic leukemia, when hemorrhages occur, can be differentiated by the general lymphatic enlargement and by examination of the blood.

7. Obesity

Definition.—Obesity is a condition of the body in which the proportion of fat to the body weight is in excess.

Etiology.—Sometimes the condition is due to an actual fault in metabolism. In other cases it is due to overeating and lack of exercise, so that though the act of metabolism may be normal it is overtaxed and there is a deposit of fat in excess of normal. The persistence of overeating and lack of exercise will surely bring about a condition which cannot be distinguished from an original metabolic fault. *Heredity* and *age* both play a rôle in acquiring obesity.

Characteristic Symptoms.—*The condition, when due to overeating,* and when the act of metabolism is normal, is characterized by deposits of fat in all portions of the body, superficially and about the intestinal organs. After a time the patient becomes indolent, dyspneic on exertion, the latter being due to fatty deposits about the heart.

In the progressive condition or where there is faulty metabolism, the patient is pale, extremely fat, overweight, and is dyspneic; there is palpitation of the heart. The urine is of rather low specific gravity; there is a tendency to albuminuria with tube casts; there may be actual edema of the body; the blood pressure is high.

Anders distinguishes between plethoric and anemic obesity. Certainly, as he states, there is no well-defined line of demarcation between the two classes, the first class being due to an individual fault, the patient often having the appearance of perfect health—florid with the blood above normal, amounting to a mild polycythemia. This individual unless he changes his habits will assuredly pass into the stage at which some are found at the beginning of the disease—pale, anemic, breathless, with high blood pressure, and with great masses of fat internally and externally.

Conditions to be Differentiated from Obesity

The diagnosis must be made between:

Myxedema
Adiposis dolorosa
Adiposis cerebralis
Adenolipomatosis.

MYXEDEMA.

In this condition the body enlarges; there is breathlessness, also mental torpor much in excess of that of obesity. The thyroid gland is atrophied, the skin harsh, dry, thick and entirely different from the soft pliable skin over masses of fat in obesity. The heart is often rapid and feeble; there is mental depression and loss of memory. The administration of *thyroid extract* acts like magic in myxedema. A heavy, dull, expressionless individual, with rapid feeble heart will in the space of a few days become an almost normal individual; no such change is noted in obesity. The patient may lose weight under the administration of thyroid extract, and even become less dyspneic because of the disappearance of the fat, but the rapid change, the appearance of an entirely new personality which occurs when a case of myxedema is put upon thyroid extract is characteristic of the latter condition.

ADIPOSIS DOLOROSA.

Adiposis dolorosa (*Dercum's disease*) is rare.

It is characterized by the appearance of symmetrical, exquisitely painful tumors in various parts of the body, but not upon the hands or feet; the skin is not involved; there is the sensation of lobulation given to the fatty masses; there is no sweating, but a possible involvement of the nerve trunks. This condition is doubtless closely related to an impairment of activity in the pituitary gland.

Adiposis cerebralis.

The obesity which accompanies cerebral conditions, such as tumors of the pituitary gland, is characterized by the infantile condition of the individual, the loss of hair, or poorly developed hair, on the individual, and the marked lack of development of the sexual organs.

8. Lipomatosis

Certain cases of Dercum's disease, according to Lyon (*Arch. Int. Med.*, volume 6, pp. 28-120) may be divided into:

1. **Adiposis dolorosa.**—This is a disorder characterized by irritating symmetrical deposits of fatty masses in various portions of the body, preceded or attended by pain and associated sometimes with asthenia and psychical changes. Cushing's studies would at least suggest that this condition is one due in many instances to some disturbance of the hypophysis. Certainly many of the cases have many of the characteristics of hypophysis disease.
2. **Nodular Circumscribed Lipomatoses.**—These are simply multiple fatty tumors in various portions of the body. At times they resemble Dercum's disease to a certain extent.
3. **Diffuse Symmetrical Lipomatoses of the Neck.**—In these cases there is a high collar of fat about the neck only, or it may be in other small areas besides. Sometimes the tumor disfigures the individual considerably, but it does not interfere with the health. The name adenolipomatoses has been given to these because scattered through the fatty masses are nodules of lymphatic tissue.
4. **Cerebral Adiposity.**—This is unquestionably a form of pituitary disease and is characterized by tumors of the hypophysis, and as described in diseases of the pituitary body, Cushing has proven beyond doubt that it belongs in this class of conditions.
5. **Pseudolipomatoses.**—These occur in hysterical individuals. They are sometimes blue and sometimes white in appearance. They appear to be infiltrated under the skin, and in many instances are true lipoma.

Conditions to be Differentiated

The diagnosis in these cases is usually quite easy. Perhaps the cerebral adiposis and possibly the adiposis dolorosa might be mistaken for myxedema, but the difference is that in myxedema there is a curious infiltrated condition of the skin, which is absent in adiposity; there is the distinct mental failure, and above all there is the marked response in myxedema to therapeutic use of the thyroid gland; it has no effect at all on cerebral adiposity. The tumors of course might be mistaken for new growths of other varieties, but careful examination will show that they are all in the subcutaneous tissue, and that they have the characteristic puckering of the surface when the skin is made tense over the surface.

9. Hemochromatosis

Etiology.—This is a curious disease of which few cases have been described. Latest investigations fairly well prove that the condition is a primary disease of the blood. The corpuscles disintegrate and give up their hemoglobin; the iron pigments are deposited in the skin; the hemosiderin is deposited in the cells of the liver, pancreas, lymphatic and sweat glands; the hemofuscin is deposited in the stomach, intestines, urinary bladder and ureter (Futcher). In the early stages there is no sugar in the urine, but it is present later.

The cases of hemochromatosis which are accompanied by diabetes, have all the symptoms of that disease plus bronzing of the skin, extremely progressive weakness and enlarged liver. The color of the skin varies from a dark brown to a bluish black.

Cirrhosis of the liver and the pancreatitis which occur are believed to be due to a common cause.

Conditions to be Differentiated from Hemochromatosis

This condition can perhaps only be mistaken for:

Addison's disease

Pigment which occurs in many other conditions, such as overuse of arsenic, or filthy pigment, etc.

The presence of enlarged liver and sugar in the urine of course mark the case as one of hemochromatosis. Pigmentation without enlarged liver and diabetes cannot be so diagnosed.

10. Ochronosis

Etiology.—Ochronosis is a disorder of metabolism, the chief symptoms of which are blackening of the cartilages and the appearance of dark colored urine due to alkapton or to derivatives of carbolic acid (Osler).

So far as is known there are cases which appear to be congenital and others which come from the prolonged use of carbolic acid.

The patient seems in perfect health, though a curious waddling gait is reported by Osler.

Diagnosis.—The mere appearance of dark urine, which may be the result of prolonged taking of preparations of carbolic acid and of phenol-salicylates, must not be mistaken for ochronosis, unless it is accompanied by pigmentation of the cartilages.

The superficial cartilages show black through the thin skin.

There is no other condition for which the disease can be mistaken.

Section V

Diseases of the Digestive System

A. Diseases of the Mouth

1. Stomatitis

Stomatitis is an inflammation of the mucous membrane of the mouth, affecting the tongue, the lips or the buccal mucous membranes.

Stomatitis has been divided into various groups, as follows:

(*a*) *Aphthous Stomatitis*

This is really a vesicular stomatitis, because in the beginning there is a true vesicle of the mucous membrane. The covering of the latter is thrust off and then a rather shallow ulcer is formed with a white base and red edges. These are most common on the edges of the tongue and on the inside of the lips. These ulcers are extremely painful; there is much salivation. Frequently there is derangement of the digestion with these cases, and occasionally there is quite sudden fever, particularly in young children.

Diagnosis.—The extreme tenderness of these ulcers and their isolated nature characterize them. They might be possibly mistaken for mucous patches, but mucous patches occur farther down in the tonsils, as a rule; further than this, there is always the history of SYPHILIS, as the deciding factor.

(*b*) *Ulcerative Stomatitis*

Ulcerative stomatitis is a more severe form. Here the inflammation begins on the margin of the gums; they become swollen and bleed, and around the edges of the gums there is often a marked membrane. The eruption spreads very rapidly; sometimes the condition occurs in epidemics.

Diagnosis.—Perhaps the only condition this could be confused with is PYORRHEA ALVEOLARIS; however, in pyorrhea there is no active inflammation, nor the same tenderness, nor the constitutional depression peculiar to this condition. Pyorrhea alveolaris is the result of inflammation of the root of the tooth.

(*c*) *Thrush—Parasitic Stomatitis*

Thrush, or parasitic stomatitis, is the result of inflammation due to a fungus, Oidium albicans. This may cover the entire interior of the mouth; it is particularly common on the tongue, on the buccal mucous membrane and on the uvula.

Diagnosis.—The diagnosis can be made absolutely by examination of the exudate, when the spores and the mycelium can be seen. This condition might be mistaken for APHTHOUS STOMATITIS; it occurs more frequently, however, in children and in those recovering from an exhausting disease. *A microscopical examination will make the diagnosis positive.*

Extensive proliferation of the exudate on the soft parts might be mistaken for DIPHTHERIA. The exudate of diphtheria, however, is markedly adherent to the mucous membrane, while Oidium albicans easily strips off. The microscope will show diphtheria bacilli in the diphtheria exudate, while it will show the fungus in the parasitic exudate. The color of the oidium is pearly white, while that of diphtheria is yellowish white or grayish.

(*d*) *Mercurial Stomatitis*

Definition.—Mercurial stomatitis is the result of long-continued small doses or an overdose of mercury, or the effect of having worked in mercury.

Symptoms.—The first symptom is hypersalivation. This is rapidly followed by swelling and tenderness of the gums, and sometimes by loosening of the teeth. In connection with these symptoms the salivary glands are frequently enlarged and tender, and in rare cases the bones of the jaw become necrosed. Salivation, however, occurs in so few conditions, that the appearance of the symptoms above detailed practically make the diagnosis.

Diagnosis.—About the only conditions with which this could be confounded are one of the OTHER FORMS OF STOMATITIS with considerable salivation, and SCURVY. In stomatitis there is more serious inflammation of the underlying mucous membrane, especially in ulcerative stomatitis and in noma. In scurvy there are distinct hemorrhages into the gums, and often into other mucous membranes; there is the history of improper diet, and often of privation. In both of these conditions there is no history of the patient having taken mercury—the deciding factor.

(*e*) *Noma—Gangrenous Stomatitis*

This condition is characterized by a rapid destructive gangrenous inflammation of the cheek. It occurs commonly in persons of poor physical condition who are the subjects of some infective disease, such as measles or typhoid fever. The condition begins with a slight ulcer on one or the other buccal surfaces; rapid induration occurs, followed in a short time by a destructive inflammation which often ends in gangrene. The whole cheek may be destroyed.

Diagnosis.—Except in the beginning, it is impossible to confound this condition with any other inflammation of the mouth. At that time it might be mistaken for a SIMPLE APHTHOUS OR ULCERATIVE STOMATITIS, but in these the condition is not surrounded by induration, and does not progress rapidly, as in noma.

As the actual cause of this condition is not known, an important point in the diagnosis is that one consider the condition possible, so as not to allow a serious inflammation of the cheek to arise before treatment is instituted.

2. Fetor oris

(Bad Breath)

Fetor oris, or bad breath, is common in all ages, and has many different causes. By the laity it is almost always thought to be due to a deranged condition of the stomach, which is perhaps the rarest of all the causes. There are certain conditions of heavy, acetonelike breath due to acidosis accompanied by symptoms of indigestion, particularly in children, but these are so characteristic that they need little description.

In certain cases of marked gastric dilatation, particularly if there is malignant ulceration, there is a foul odor which is therefore due to actual derangement of the stomach.

Most of the bad breaths are due to the following causes: *

First.—Nasal conditions and dry atrophic ozena, which give rise to a sickening odor of the breath.

Second.—Retained matter in the follicles of the tonsils. This can be discovered by pressing an instrument against the tonsil, and noting the odor, which is of a horrible, fetid characteristic nature.

Third.—Pyorrhea.—The suppuration about the roots of the teeth gives rise to an odor which is unmistakable and well known. This can be discovered by cleaning the base of the teeth and noting the odor on the instrument used.

Fourth.—Any condition of the mucous membrane of the mouth and pharynx, such as the various forms of stomatitis—especially ulcerative and gangrenous stomatitis—are fertile sources of fetor oris.

Fifth.—Certain conditions of the lung, especially gangrene, and large cavities in tuberculosis and bronchiectasis, give rise to most unpleasant odors to the breath. This type is characterized by the peculiar sweet gangrenous odor common to gangrene in any portion of the body.

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* Arrangement of Osler.

3. Leukoplakia

Leukoplakia is characterized in the beginning by deep red, erythematous patches (Reisman) upon the mucous membrane of the cheeks, gums and tongue, later becoming pearly white, and having the appearance which comes from the application of silver nitrate. These patches may vary in size from a millet seed to patches covering almost the entire mucous membrane of the lips and gums.

This affection occurs sometimes as the result of syphilis, and is very common in smokers. It has a tendency, under certain conditions, to become malignant.

Conditions to be Differentiated from Leukoplakia

It must be distinguished from:

Parasitic stomatitis
Diphtheria and other acute conditions
Epithelioma.

PARASITIC STOMATITIS.

Leukoplakia can easily be distinguished from parasitic stomatitis by a microscopic examination, the germ being found in thrush and not in leukoplakia. Again the exudate of thrush is not closely connected with the mucous membrane, and can easily be scraped off. The white patches of leukoplakia cannot be detached.

DIPHTHERIA.

Diphtheria and other acute conditions can be recognized by the presence of their systemic symptoms—fever, prostration and so forth, and by the specific germ of the conditions—all these are wanting in leukoplakia.

EPITHELIOMA.

Epithelioma must be separated by the facts that in malignant disease there is a thickening due to infiltration of the tissues, and often also enlargement of the lymphatic glands. This is not the case in leukoplakia. It must not be overlooked that leukoplakia occasionally takes on a malignant degeneration; hence any case of leukoplakia with suspicious infiltration, or a patch which becomes degenerated, must at once be treated as a malignant condition.

4. Geographical Tongue

Geographical tongue is a condition which is the result of rapid exfoliation of the epithelium of the tongue in irregular sinuous patches. The affection has few symptoms. Its curious bright red patches of irregular shapes intermingled with the normal coat of epithelium are quite characteristic.

Conditions to be Differentiated from Geographical Tongue

It may, however, be mistaken for:

Inflammation of areas of the tongue
Stomatitis
Syphilis.

INFLAMMATION OF AREAS OF THE TONGUE—STOMATITIS.

The red patches of geographical tongue are smooth and not painful, and do not give rise to an exudate as do the areas affected by stomatitis.

SYPHILIS.

The marked contrast between the areas of coat and desquamation might lead to a diagnosis of syphilis, but there are none of the other signs of syphilis such as skin lesions, enlarged spleen, snuffles, sore throat, etc.

B. Diseases of the Salivary Glands

1. Xerostomia

(*Arrest of the Salivary and Buccal Secretions, Dry Mouth*)

Xerostomia is a condition of dryness of the mucous membrane of the mouth, which occurs frequently in women and in nervous individuals. The mucous membrane is dry, sometimes cracked, and gives rise to a great deal of discomfort; sometimes mastication and swallowing are interfered with.

Diagnosis.—It may easily be distinguished from the various forms of STOMATITIS described, but might be mistaken for the DRY MOUTH, which is present in DIABETES. From the latter it of course can be distinguished by examination of the urine where sugar is present, and its absence noted in xerostomia.

The EFFECTS OF ATROPIN might be mistaken for the condition, but this is temporary and acute, while xerostomia is a chronic condition.

2. Oral Sepsis

Hunter has called attention to the fact that severe anemias have their origin in septic conditions of the mouth, particularly in diseased conditions about the roots of the teeth, pyorrhea alveolaris being the most fertile source. It is certain also that other conditions besides anemia have their origin here—arthritis, endocarditis and other serious states are common sequelae of pyorrhea. Chronic tonsillitis and buried and unerupted teeth have been found to be the actual causative factor in many prolonged arthritic conditions.

Diagnosis.—The evident point in the diagnosis is, *care in general examination* of the patients. Pyorrhea, alveolar abscesses and chronic tonsillitis can be diagnosed at sight. It remains to trace the relation between such observed conditions and the diseased state under observation. It must always be remembered that oral sepsis and arthritis may be only relative.

3. Inflammation of the Salivary Glands

Etiology.—Inflammation of the salivary glands can occur in ptyalism or in the specific parotitis. (See Parotitis.) It may occur as the result of mouth infection, particularly in the course of the infectious fevers. A suppurative inflammation, especially of the parotid glands, is particularly prone to occur after abdominal operations and after dysentery. It also may result from injury to the urinary tract or the alimentary canal.

A chronic inflammation of all the salivary and lacrimal glands is known by the name of *Mikulicz's disease.*

These various inflammations of the salivary glands are simply symptomatic. Careful search must be made in the diagnosis for the existing cause, be that scarlet fever, diphtheria, or any other infection.

Diagnosis.—These inflammations cannot be mistaken for any condition except Ludwig's angina and inflammation of the lymphatic glands.

In LUDWIG'S ANGINA the inflammation often simulates, and, indeed, it is accompanied by enlargement of the submaxillary salivary glands, but here again the distinction is easily made because of the rapid increase of the inflammation in these parts in Ludwig's angina. The septic condition and high fever of the patient are characteristic of Ludwig's angina.

Inflammation of lymphatic glands can be distinguished from the position of the swellings.

C. Diseases of the Pharynx

1. Hyperemia of the Pharynx

This condition is characterized by redness, slight swelling, at times dryness, and mucous exudate on the pharynx, and is often the result of inhalation of irritating substances such as tobacco and hot vapors. It is constantly present in the infectious diseases such as tonsillitis, an ordinary cold, scarlet fever and diphtheria. Singers and speakers are often affected.

Diagnosis.—The important point of the diagnosis is, not to mistake a simple hyperemia due to slight infectious and irritating substances, such as smoking, for the more severe HYPEREMIA DUE TO infectious diseases like SCARLET FEVER AND DIPHTHERIA. These latter may be differentiated by their peculiar symptoms and also in diphtheria particularly, by a bacteriological examination of the exudate. There may be a passive HYPEREMIA DUE TO STASIS OF THE VESSELS FROM HEART DISEASE. This of course can be diagnosed by the presence of the dilatation of the right heart. In certain cases of heart disease, such as aortic regurgitation, the pulsation may be seen in the vessels and the whole of the uvula may be seen to pulsate.

2. Hemorrhage of the Pharynx

Hemorrhage, as the name indicates, is due to extrusion of blood from the pharyngeal surface, enlarged vessels due to varices, and due to engorgement from cardiac conditions and ulceration—all are the causes of this hemorrhage.

Diagnosis.—It must never be confounded with spitting of blood due to NOSE-BLEEDING, in which the blood comes from the posterior nares of the pharynx, or to spitting of blood due to TUBERCULOSIS of the lungs. A safe rule to follow is that blood spitting from the mouth, particularly in the act of coughing, when not the result of an evident lesion of the gums or a local lesion of the pharynx, is due to a condition of the lungs. It is rare indeed that hemorrhage from the pharynx gives rise to sufficient bleeding to be called "spitting of blood," and the diagnosis must depend of course upon the local condition found, upon the condition found in the lungs and upon the history of the case.

3. Edema of the Pharynx

This condition is the result of irritating materials, sometimes of nephritis, or the result of severe inflammatory throat conditions, such as quinsy, postpharyngeal abscess and diphtheria. The swelling is characteristic and can be mistaken for nothing else. It is boggy, has the appearance of a gelatinous material, the important point being to discover the cause. This can be done by searching for nephritis and for local inflammatory conditions which will interfere with the circulation.

4. Acute Pharyngitis

Acute pharyngitis is simply an increased condition of hyperemia, and is the result of some local inflammation or irritation continued longer than a few hours. It is present in all the acute infections, such as measles, scarlet fever and diphtheria. Occasionally it is the result of an infection which simply affects the nasopharyngeal mucous membrane, such as coryza. At times it is accompanied by deafness due to obstruction of the eustachian tubes, which may be due to swelling of the mucous membrane. Here again the important point is to find the *cause of the inflammation.*

5. Chronic Pharyngitis

Chronic pharyngitis is present in sore throats due to long-continued tobacco smoking, which brings about a chronic nasopharyngeal catarrh, and is often characterized by a glazed appearance of the mucous membrane and also by enlargement of the pharyngeal lymphatic glands. This enlargement is quite characteristic, and during an acute attack of pharyngitis the pharyngeal lymphatic glands often are the seat of a yellowish exudate.

6. Ulceration of the Pharynx

Ulceration of the pharynx may be an acute inflammation of the chronic ulceration spoken of above, in which case the ulcers are red, swollen, and covered with an exudate, and are only a part of an acute pharyngitis. Simple ulceration is due to an extension of aphthous stomatitis and to the action of corrosive substances; syphilitic ulcerations are due to syphilitic infections, and are deep-seated and eroded. Ulceration due to tuberculosis may also be present.

Differentiation.—These various ulcerations may be differentiated—syphilitic forms may be recognized by the presence of syphilitic lesions in other portions of the body; if this is not evident a Wassermann reaction should be employed. Tuberculous ulcerations of the pharynx are rarely primary, and are almost always the result of a tuberculous condition of other portions of the body and must be diagnosed thereby and by recovering tubercle bacilli from them. Occasionally ulceration of the pharynx occurs as the result of diphtheria and typhoid fever. The presence of these two diseases will make a positive diagnosis as to the character of the ulceration.

7. Retropharyngeal Abscess

Retropharyngeal abscess is often the result of caries of the vertebra, and occasionally is due to a deep inflammation resulting from a superficial inflammation such as scarlet fever or diphtheria.

There is difficulty in swallowing, extreme pain, and if a child, the patient cries constantly. Examination of the mouth will reveal a bulging pharynx, and palpation with the finger will discover a fluctuating tumor.

Differentiation.—It is important to differentiate between the ABSCESS DUE TO SUPERFICIAL INFECTIONS and THOSE DUE TO CARIES OF THE SPINE, on account of the entire difference in treatment. This of course must be done by excluding caries of the spine through examination of the cervical region. In caries there is tenderness and fixation, and extreme pain on moving the neck. This is entirely absent from abscess due to local infection. An x-ray will demonstrate a disease of the bones of the spine, if present.

8. Ludwig's Angina

(*Angina Ludovici, Cellulitis of the Neck*)

Ludwig's angina is a deep-seated streptococcic infection beginning often in the neck. The condition is characterized by pain, extreme difficulty in swallowing, very rapid inflammatory induration of the entire side of the neck, so that swallowing and breathing may be impeded.

Diagnosis.—The rapidity of the process, and the very evident sudden, severe illness of the patient, together with brawny induration of the skin and underlying tissues will distinguish this from a SIMPLE INFLAMMATION OF THE GLANDS; the latter is more localized, and soon gives rise to suppuration, whereas Ludwig's angina rarely suppurates early enough to distinguish it without an operative procedure.

D. Diseases of the Tonsils

Follicular Tonsillitis

This is an acute inflammation of both tonsils; its onset is sudden.

Symptoms and Physical Signs.—There is sore throat, fever—often reaching 103° to 104° F.—chilly sensations or an actual chill, severe headache and aching limbs.

Both tonsils become red and swollen, the follicles being the seat of a yellowish pultaceous exudate. At times the whole tonsil is covered with the exudate which has spread over the surface from the follicles. It is easily removed, leaving no bleeding surface. A culture of this exudate shows a growth of either a staphylococcus or streptococcus.

Conditions to be Differentiated from Follicular Tonsillitis

Follicular tonsillitis is to be differentiated from:

Scarlet fever
Diphtheria
Vincent's angina.

SCARLET FEVER.

Here there is the same high fever as in acute tonsillitis, but the child seems more seriously sick. Within twenty-four hours the characteristic rash appears. The exudate in the throat is not confined exclusively to the tonsils.

DIPHTHERIA.

Here, as described in the article on diphtheria, the throat is not so painful; the fever may not be so high; the exudate is a true membrane leaving a bleeding surface behind; it is not confined to the tonsils. A culture will show diphtheria bacilli.

VINCENT'S ANGINA.

Vincent's angina is characterized by the following symptoms: the tonsils are affected; there is a peculiar fetid odor to the breath; there is a punched-out ulcer. A fusiform bacillus and a spirillum may be seen in a smear from the throat.

2. Suppurative Tonsillitis

(*Quinsy—Peritonsillar Abscess*)

This is a condition due to infection of the tonsils. It may involve only the tonsillar tissue itself, or it may include the peritonsillar tissue as well.

Characteristic Symptoms.—It is characterized by swelling of one or both tonsils, by edema of the surrounding parts and by great difficulty in swallowing, it frequently being impossible for the patient to swallow his saliva on account of the great pain. Occasionally the disease begins as an ordinary follicular tonsillitis, the infection being due to the organism which causes the latter disease—usually the staphylococcus or streptococcus. The infection soon extends to the peritonsillar tissue; pain and fever become extreme. When the abscess which forms is peri- or retrotonsillar, it points in the soft palate just above the posterior molar tooth. This pointing and suppuration can be discovered by inserting the finger in the mouth, putting the other on the outside of the neck and making a bimanual examination.

Conditions to be Differentiated from Suppurative Tonsillitis

The condition must be differentiated from infections due to *other conditions than suppuration* of the tonsils and peritonsillar tissue. It must be differentiated from:

Follicular tonsillitis
Diphtheria
Scarlet fever
New growth or leukemic enlargement of the tonsil.

FOLLICULAR TONSILLITIS.

In follicular tonsillitis, which it resembles and from which it occasionally results while the tonsils are swollen, there is not much tenderness, and the difficulty in swallowing is not nearly so great as in suppurative conditions. There is no formation of pus.

DIPHTHERIA—SCARLET FEVER.

From diphtheria and scarlet fever it must be differentiated by the fact that in the latter the swelling affects the neck proper and the lymphatic glands surrounding the tonsil region. The presence of suppuration is indicated by pointing, which can be discovered by manipulation with the finger—is positive proof that suppuration is going on. Often following scarlet fever and diphtheria there is suppuration in the neck, but usually this is the result of infection and breaking down of one or more of the lymphatic glands, and does not have the same position as does a true peritonsillar abscess which points in the throat usually just above the lower molar teeth.

A NEW GROWTH OR LEUKEMIC ENLARGEMENT OF THE TONSIL.

These conditions might possibly be mistaken for suppurative tonsillitis, but the acute inflammatory symptoms are wanting except in rare instances. The author has seen a case of acute lymphatic leukemia with rapid painful inflammation of the tonsil, but the blood count easily made the diagnosis.

3. Chronic Tonsillitis

Etiology.—Chronic tonsillitis is the result of overgrowth of both tonsils, continuing over months and years. This usually results from repeated acute attacks of tonsillitis, although it sometimes seems to be present in individuals who have not suffered from these attacks and in whom the lymphatic tissue over the body is redundant.

Characteristic Features.—It is almost always accompanied by increase in the size of the pharyngeal tonsil giving rise to the so-called "adenoids." In these individuals there is almost always mouth breathing, there is a thick nasal tone to the voice; in extreme cases the patient is not developed properly, he has a short upper lip, and may have a prominent sternum with a Harrison groove at the base of the chest. These cases must be carefully examined for any other condition. The condition is marked by its chronicity and its interference with breathing.

Large tonsils and adenoids, when they give symptoms, should be referred to a surgeon, but there are a great many cases where surgical interference is not necessary.

Differentiation.—This condition can be confused with other conditions which cause mouth breathing and nasal tone to the voice. CLEFT PALATE gives such a voice, but examination of the throat will show at once that the tonsils are not involved. Nasal obstruction from polyps, enlarged turbinated bones and deflected septum may cause both mouth breathing and a nasal tone to the voice, but careful examination will easily make the difference between the two conditions evident.

4. Vincent's Angina

Origin.—This is an infection of the throat, usually beginning in the tonsil. The organism to which the disorder seems to be due is a fusiform bacillus (Vincent's bacillus) and a spirillum.

Characteristic Features.—The attack usually begins slowly, and is somewhat prolonged in its course. It may, however, have the characteristics of any acute infection, headache, fever, and sore throat being the most common symptoms. In the light and acute cases the attack lasts only a few days, but in the severe cases it may be prolonged for several weeks.

The throat condition characteristic of the disease usually begins on one tonsil, though both may be affected. The patch is grayish white and has a tendency to run over the soft palate toward the uvula. The gums may also be the seat of the exudate.

In removing the membrane, which is not so tough as in diphtheria, a bleeding surface is left. Often on removal of the membrane the lesion is found to consist of a more or less deep ulcer which has been obscured by this exudate.

Differential Diagnosis.—The differential diagnosis can only be made by a smear and staining. Neither the fusiform bacillus nor the spirillum will grow on the ordinary culture medium.

Conditions to be Differentiated from Vincent's Angina

The condition is in appearance much like:

Diphtheria or
Follicular tonsillitis.

DIPHTHERIA.

Diphtheria is at once distinguished by the presence of the Bacillus diphtheriæ which can be seen in a smear from the throat, or will grow on the culture medium. The symptoms of diphtheria too are more severe as a rule; there is glandular involvement and more prostration. The punched-out ulcers common in Vincent's angina are not present in diphtheria.

FOLLICULAR TONSILLITIS.

This is more acute as a rule than Vincent's angina; the exudate is likely to be in the follicles of the tonsils; there is not the punched-out shape of the ulcer.

The fusiform bacillus and the spirillum of Vincent are wanting in the smear.

E. Diseases of the Esophagus

1. Acute Esophagitis

Cause.—Acute esophagitis may be the result of swallowing corrosive liquids, such as acids, or it may be the result of infections from acute fevers, or the result of traumatism (swallowing some hard large object) or it may be spontaneous in nursing children.

Symptoms.—Under all these circumstances the symptoms are difficult and painful swallowing; there is always pain underneath the sternum, depending upon the degree of inflammation. Blood or mucus may be vomited or swallowed.

Conditions to be Differentiated from Acute Esophagus

In esophagitis there is always the history of one of the above causative factors. It can be mistaken for:

OBSTRUCTION, the result of some outside condition, such as pressure from a new growth; also from cancer or other stricture of the esophagus.

A NEW GROWTH OUTSIDE OF THE ESOPHAGUS, SUCH AS AN ANEURISM OR MEDIASTINAL GROWTH, can be diagnosed by the signs of those conditions.

CANCER OF THE ESOPHAGUS, OR ANY OTHER NEW GROWTH INVOLVING

THE ESOPHAGUS, has actual obstruction of the tube as one of its characteristics. This does not exist in simple esophagitis.

2. Ulceration of the Esophagus

Ulceration of the esophagus occurs again as the result of infections such as diphtheria. True peptic ulcers may occur as the result of regurgitation of gastric juice into the esophagus through the cardiac orifice; these ulcers are characterized by pain and by difficulty in swallowing. Perforation has occurred in peptic ulcer. Such a case was brought under the author's care; it occurred in a hysterical woman with an original esophagismus, or perhaps cardiospasm. Perforation occurred as the result of passing a bougie, the latter perforating one of the ulcers. At autopsy, the entire mucous membrane was found riddled with small punched-out ulcers, ranging in size from a pin's head to one-quarter of an inch in diameter. This case was reported by Dr. John Guiteras, who made the pathological examination.

Conditions to be Differentiated from Ulceration of the Esophagus

These ulcerations can be distinguished from SIMPLE ESOPHAGISMUS by the longer duration of this condition and by the presence of an hysterical history in the case, the result usually of regurgitation of gastric juice.

ULCERATIONS WHICH OCCUR AS THE RESULT OF NEW GROWTHS must be distinguished by recognizing a new growth by its obstruction and other signs.

3. Esophageal Varices

Esophageal varices occur as the result of a dilatation of the veins at the gastric end of the esophagus. This gives no symptoms until a rupture occurs, the first being either vomiting of blood or passing of large amounts of blood from the bowel, the patient often becoming syncopal after the vomiting; there is no pain. These veins are enlarged in cirrhosis of the liver, and in a few cases of heart disease.

Diagnosis.—The diagnosis must depend upon the exclusion of GASTRIC ULCER, CARCINOMA OF THE STOMACH, and on the presence of CIRRHOSIS OF THE LIVER, or extensive HEART DISEASE. For the signs present in these conditions, reference may be made to the proper article.

4. Rupture of the Esophagus

Etiology.—Rupture of the esophagus sometimes occurs by traumatism due to undue violence in passing a sound. It is said to occur as a result of violent vomiting.

Diagnosis.—The diagnosis of the rupture must depend upon the violent vomiting, and the local inflammation in neighboring organs, the latter giving rise to pneumonia, pleurisy, or mediastinitis.

Given the history of the passing of a bougie, followed by a sudden onset of pneumonia, pleurisy or mediastinitis, scarcely any mistake can be made.

5. Dilatation and Diverticula

Etiology.—Dilatation and diverticula of the esophagus occurs as the result of strictures of one sort or another, or the diverticula may be congenital, there being great dilatation and hypertrophy.

Diagnosis.—Both these conditions may be diagnosed positively by the x-ray, a bismuth meal being administered and a picture taken afterward. The diverticulum may be mistaken for a stricture itself because the sound may pass into the pouch and be unable to go further. Under these circumstances an x-ray is always demanded.

6. Esophagismus

This is a spasmodic condition of the esophagus which prevents food from being properly swallowed. It occurs in hysterical individuals.

Diagnosis.—The diagnosis must be made by attempting the passage of a stomach tube, which will usually be hindered from passing, but if a solid bougie is passed, it enters the stomach without difficulty.

The differential diagnosis must be made between esophagismus and CARDIOSPASM; the latter, as will be noted further on, is the result of spasm of the cardiac end of the stomach and not of the esophagus.

The diagnosis should be influenced by the position in which the contraction occurs. An esophagismus does not give rise to dilatation of the esophagus above the part affected by the spasm; this however is the case in cardiospasm. If there is any dilatation above the cardia in cardiospasm a sound may be difficult to pass into the stomach, but this can be overcome by the use of Plummer's sound, where a thread is swallowed and then passed through an opening in the sound. The thread being fixed below, because it extends far down the gut, gives an easy method of directing the sound over a small dilatation.

7. Stricture of the Esophagus

Stricture of the esophagus is a narrowing of the tube, due to the following various causes: (*a*) to a formation of connective tissue, the result of ulceration from corrosive poisons; (*b*) to syphilis; (*c*) to involvement of the walls of the esophagus by malignant growths; (*d*) to an old and severe ulceration not due to corrosive poison, or (*e*) it may be due to pressure from without, such as an aneurism, mediastinal growths or tuberculous glands.

Conditions to be Differentiated from Stricture of the Esophagus

It must be differentiated from:

Esophagismus
A diverticulum
Cardiospasm.

The diagnosis is established by the inability of the patient to swallow, and this is confirmed by the lack of power to pass a sound.

The *diagnosis of the character of the stricture must depend entirely upon the history of the case.*

ESOPHAGISMUS.

It is of the greatest importance to distinguish it from esophagismus just referred to in the previous article; this may be done, as stated, by the use of a stiff sound, instead of a soft tube. The diagnosis may be confirmed by the use of a bismuth meal followed by an x-ray examination.

DIVERTICULUM.

In a diverticulum the best possible diagnostic method is the use of a bismuth meal. This will show a shadow in the exact place occupied by the diverticulum.

CARDIOSPASM.

In cardiospasm a soft tube cannot be passed, but by the use of a Plummer's sound which is threaded, the obstruction can usually be overcome. (See Cardiospasm.)

F. Diseases of the Stomach

1. Acute Gastritis

Etiology.—Acute gastritis is an acute catarrhal inflammatory condition of the mucous membrane of the stomach, due usually to errors of diet or, possibly, to some corrosive substance that has been swallowed.

Characteristic Features.—The characteristics of the disease are pain in the epigastrium, nausea, vomiting and distaste for food. The case comes on acutely or merges gradually into the more subacute condition of ordinary indigestion. The pain may be severe, or it may be simply a sensation of pressure.

Conditions to be Differentiated from Acute Gastritis

The following conditions are constantly mistaken for this:

Gall-stone colic
Appendicitis
Gastric or duodenal ulcer
Angina pectoris
Gastric crises of locomotor ataxia
Toxemia of pregnancy
Acute pancreatitis.

There is no excuse for confusing these conditions with acute gastritis; nevertheless, it is constantly done. It occurs in the following way: In the first place the vomiting and pain in the epigastrium which frequently accompany all of the above conditions are often considered as the result of an acute gastritis, without sufficient care being taken to exclude a more serious affection. Again if the stomach contains food, particularly if the food is undigested, and is vomited in the beginning of an attack of appendicitis, gall-bladder disease, or pancreatitis, the presence of the undigested food may be taken as sufficient evidence of an ordinary attack of indigestion, and this may deter the patient from calling a physician or the physician may fail to make a careful examination.

Again infections like scarlet fever and pneumonia are sometimes looked upon as simply acute gastritis, because of the vomiting with which they are so frequently ushered in. This error can be recognized by the fact that the symptoms and physical signs of the causative disease are soon manifest.

GALL-STONE COLIC.

From gall-stone colic, gastritis may be distinguished by the fact that the pain and distress from acute gastritis are usually relieved either by limitation of the food, by vomiting the food already taken, or by lavage. This is *not* the case in gall-stone colic, for while vomiting does sometimes relieve the pain, because it relieves the accompanying spasm of the gall-ducts, it almost, without exception, will return. In gall-stone colic there may be jaundice; there almost always is tenderness over the gall-bladder. This tenderness differs from the tenderness of acute gastritis, because it is localized over the region of the gall-bladder and is not persistent over the entire area of the stomach.

APPENDICITIS.

Acute gastritis may be mistaken for appendicitis. Usually, however, in appendicitis, while the tenderness at first may be over the epigastrium, it is transferred later to the region of the appendix, and the condition is usually accompanied by leukocytosis, which is *not* present in acute gastritis. The continuation of the pain, the local tenderness and fever mark the case as one decidedly *not* acute gastritis.

Gastric or Duodenal Ulcer.

In gastric or duodenal ulcer, the pain is either aggravated after ingestion, or if long after, it is relieved by taking food. This distinct history of the case of pain coming on regularly before eating or two or three hours after, and being relieved by food, and the fact that the attacks occur periodically, often in the spring and fall, mark the condition as other than acute gastritis. The presence of blood in the vomitus and stools will also distinguish the case. A careful x-ray examination of the gastro-intestinal tract will usually indicate an ulcer if it is present.

Angina pectoris.

Angina pectoris is constantly mistaken in the milder forms for acute indigestion and is so treated. No greater mistake can be made than this. Careful inquiry will usually discover that in such cases the pain comes on *after exertion* and is instantly relieved by the patient standing still or sitting down. Examination of the cardiovascular system will usually show some marked changes in these organs which are not present in a simple gastritis.

Gastric Crises of Locomotor ataxia.

These are very frequently mistaken for acute gastritis. Here the only possible method of making a differential diagnosis is, again, careful examination of the patient. Absence of the patellar reflexes, absence of the pupil reflexes to light, irregular station or of the gait, will at once call attention to the true nature of the case.

Toxemia of Pregnancy.

Epigastric pain occurring in pregnancy as an indication of toxemia is frequently mistaken for an acute gastritis. When acute epigastric pain occurs in a pregnant woman who develops high blood pressure and albumin, the chances are the condition is *not* an acute gastritis, but a toxemia of pregnancy and should be so treated.

Vomiting of pregnancy might easily be mistaken for a local condition. This mistake is usually made when the patient is unmarried and either ignorantly or knowingly conceals her pregnancy. Constant or periodical vomiting in a healthy woman, especially if occurring in the morning, is always suspicious of pregnancy. A physical examination and the history of missed menstrual periods will make the diagnosis.

2. Chronic Gastritis

Etiology.—Chronic gastritis is the result of overeating, improper methods of eating, of eating improper food, of the abuse of alcohol, or it may be the result of chronic cardiac or renal disease. It may last for years as a condition only of inveterate dyspepsia; it may of course be secondary to some primary exciting cause.

Symptoms.—The symptoms are fullness and distress after eating, vague pains, eructations of gas and food, constipation and diarrhea; there is usually tenderness over the area of the stomach, together with heartburn. Nausea is common and vomiting frequently occurs without particular relation to food. The vomitus is characterized by retained material, by mucus, and is often of a foul-smelling acid odor. Constipation is common. Frequently there is headache and a feeling of malaise and general ill-being. There is frequently marked vertigo.

On examination, the stomach contents are shown to have a lowered acidity, and in some cases hydrochloric acid is entirely absent, however the acid may be normal or increased; mucus, leukocytes and shreds are often found.

Diagnosis.—The first step toward making a diagnosis is to take a careful history, in order to discover whether the patient eats the proper food, whether he eats it hastily, the stability of his mental condition—whether distressed about his work or domestic affairs. The next step is a careful physical examination to exclude a possible primary cause, such as nephritis or cardiac disease. Examination of the abdomen must be made in order to rule out or establish the possible presence of chronic cholangitis, gall-stone disease, chronic appendicitis and pancreatitis.

Where there is no history of nervous strain, where proper attention to the diet does not cure the condition, and where physical examination fails to discover any organic nervous disease, then one must fix one's attention upon the possibility of several conditions:

Gall-stones
Chronic appendicitis
Dilatation of the stomach
Ptosis of the stomach
Gastric ulcer
Carcinoma
Pancreatitis
Infection of the bile passages from any cause.

GALL-STONES.

Gall-stones as a rule—but by no means invariably—have a history of distinct paroxysms of pain followed by tenderness and resistance in the gall-bladder region. There may or may not be jaundice. If jaundice is extreme there is surely an obstruction of the common or hepatic duct.

Chronic Appendicitis.

Chronic appendicitis is also usually characterized by attacks of pain, periodical in character, often referred to the epigastrium, but more frequently to the region of the appendix. Frequently, and especially during a paroxysm of pain, there is leukocytosis and there is tenderness over the appendiceal region. Here a history of a previous frank attack of appendicitis will help much in the diagnosis.

Dilatation and Ptosis of the Stomach.

These conditions can be positively diagnosed by the use of a bismuth meal and an x-ray picture. If the x-ray apparatus be not at hand, then the stomach may be inflated by the use of a stomach tube and its position marked by percussion. Both conditions give clinical evidence of very marked indigestion.

Gastric Ulcer.

Gastric ulcer is more frequent in men than in women, and as a rule has a history of periodicity and chronicity. The pain is usually more severe than in gastritis; it comes on three or four hours after ingestion and is relieved by eating or the taking of an alkali. The gastric contents are acid as a rule. There may be occult blood in the stomach contents or in the stool; there is frequently a tender spot immediately in the epigastrium. *Serial x-ray plates are of the greatest value in diagnosis.*

Carcinoma.

There are usually the ordinary symptoms of chronic gastritis in the beginning of carcinoma, but persistent pain, persistent distress in an individual over fifty, with emaciation and loss of appetite, are foundation for grave suspicion of carcinoma. Here too examination of the stomach contents or stool may show occult blood. The stomach contents may contain lactic acid and Oppler-Boas bacilli. Hydrochloric acid may be reduced, but this varies. *X-ray pictures are of great value;* they show a defect in the stomach wall, a dilatation of the entire stomach, or the reverse, a small-sized stomach.

Pancreatitis.

In pancreatitis there are symptoms of ordinary indigestion with more or less epigastric pain and tenderness; there may be symptoms of gall-stones. If the stools are examined there will be found an overabundance of fat and undigested meat fibers. *The stomach contents are not affected by pancreatitis.*

Various membranes, especially the so-called Lane's kink, where the ileum is sharply angulated upon the cecum at the junction with the cecum, give rise to abdominal pain and symptoms of indigestion, which may be mistaken for chronic gastritis. However, the *gastric contents in these membrane affections are not the same in character as chronic gastritis, the contents being invariably normal.* The x-ray will give much evidence of value, showing the ileum in close contact with the iliac fossa where there is a membrane; this is not the case where there is a simple gastritis.

There is perhaps no more important and difficult task imposed upon the physician than the differentiation of these various conditions. All of us are prone to mistake appendicitis, gall-duct and gall-bladder infection, beginning carcinoma and gastric ulcer for simple chronic gastritis, until some severe and perhaps fatal complication causes us to be aware of our mistake. I believe an excellent rule is *to subject every case of chronic indigestion to all the valuable diagnostic tests, to carefully regulate their diet, to use the x-ray in every doubtful case*—and x-rays with the fluoroscope in skillful hands are becoming most useful in distinguishing all forms of gastro-intestinal conditions. *If these all fail then I believe an exploratory operation should be done.*

Cole, an earnest advocate of the Röntgen ray as a diagnostic means, and an expert of the first order, says (*Am. J. Med. Sci., July,* 1914): "Röntgenological diagnosis can be made without loss of time or strength to the patient, and indicates whether medical or mechanical treatment will suffice, or whether surgical procedure is necessary."

3. Chronic Dilatation of the Stomach

Chronic dilatation is usually the result of obstruction of the pylorus, either due to fibrous thickening, to a new growth, or to adhesions cutting off the pylorus. In these cases there is a history of preëxisting ulcer or an inflammatory condition about the gall-bladder, or of a carcinoma.

Symptoms.—The symptoms are those of a chronic indigestion, with periodical vomiting, the vomitus consisting of fermented, soured, fetid material, the result of decomposition or retained stomach contents. Tetany with all its characteristic symptoms is sometimes the result of chronic dilatation of the stomach.

Diagnosis.—The diagnosis can be made by the fact that the *individual vomits more than he appears to swallow,* that the vomiting is cumulative, that is, it comes on a long time after ingestion and the patient may be in comparative comfort between the spells of vomiting. If the condition is the result of a chronic systemic state there are simply the symptoms of chronic indigestion, as mental hebetude with eructations of foul gas and occasionally vomiting of enormous amounts of material. Physical examination in these cases shows the stomach apparently with a greater curvature down to and sometimes much below the umbilicus, with the lesser curvature about in the normal position. Sometimes, however, the stomach is ptosed as well as dilated, the size being entirely too great and the position too low.

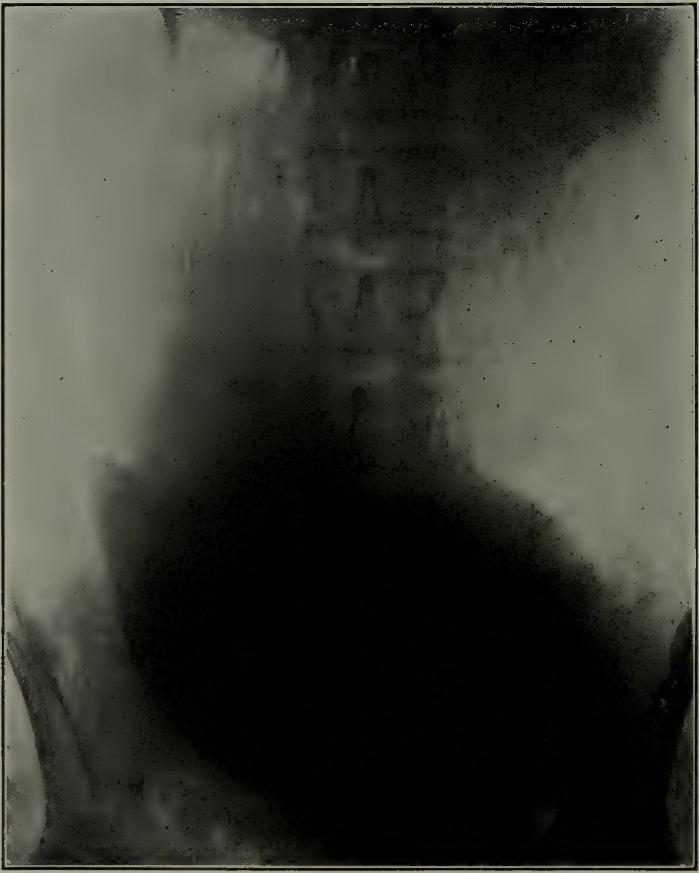


Fig. 51.—Dilatation of Stomach Due to Pyloric Obstruction from Ulcer. (Personal Observation.)

Two methods can be employed to make the diagnosis—inflation of the stomach through a stomach tube, and the use of a bismuth meal, either a fluoroscope should be used or an x-ray picture taken (Fig. 51).

Conditions to be Differentiated from Chronic Dilatation of the Stomach

The symptoms here may be confounded with the symptoms due to:

Ordinary chronic gastritis
Gastric ulcer
Gall-stone disease
Appendicitis.

It seems scarcely necessary that attention be again called to these points, but the author would put himself upon record as saying that all such cases of chronic indigestion demand the closest scrutiny, every method of diagnosis being used in order to exclude if possible any of the severe organic diseases, and in cases of doubt he would urge an exploratory operation.

The following conditions also must be differentiated:

Dilatation of the colon
Chronic gastritis
Gastric crisis
Dilatation of the esophagus.

Dilatation of the Colon.

Dilatation of the colon frequently resembles a dilatation of the stomach. Here careful x-ray examination, both with the fluoroscope and a plate, will surely make a positive diagnosis. If this means is not at hand, emptying the stomach with a tube and inflation of the colon through the rectum will help to make a positive diagnosis.

Chronic Gastritis.

In chronic gastritis due to dietetic errors, the stomach is of normal size, as proven by inflation and x-ray.

Gastric Crisis.

Gastric crisis may simulate the vomiting, but there are always the symptoms and signs of the disease if the spinal cord and the stomach are of normal size.

Dilatation of the Esophagus.

Dilatation of the esophagus causes vomiting periodically, but the vomitus is not digested; there is not the same foul odor to the vomitus. An x-ray will show the dilated esophagus.

4. Acute Dilatation of the Stomach

Mortality.—This is a condition heretofore with a very high mortality —72 per cent in 102 cases.

Etiology.—It is due to two factors, first an innervation of the stomach, causing thereby a dilatation, and secondly a constriction of the duodenum where it passes from the mesentery over the vertebral column. It is by far the most common in postoperative cases, particularly in cases where the gall-bladder or gall-ducts have been operated upon. It occurs in pneumonia, typhoid fever and some other serious diseases or it may occur without any previous disease, apparently from great error in diet or the drinking of large quantities of water; it is also probably an end result in certain cases of chronic dilatation of the stomach.

Symptoms and Physical Signs.—The symptoms and physical signs of acute dilatation of the stomach are as follows: Vomiting, abdominal pain, abdominal distention (due to enlarged stomach), constipation (diarrhea in a few cases) collapse, splashing sounds and peristaltic movements over the stomach.

Vomiting.—This is a most frequent symptom, from which there is immediate relief after lavage. It occurred in all but one of the cases reported by the author, and was present in 90 per cent of Conner's cases, from all causes. The vomitus is yellowish, greenish or blackish in color; the quantity is usually large—one pint or more—though occasionally it is small. Vomiting *per se* is painless and has much the character of that observed in general peritonitis—it is suddenly and violently expelled from the mouth, without effort on the part of the patient; it often has an actual fecal odor.

Pain is complained of in the region of the stomach. It occurs in about one-third of the cases; Conner reports 42 cases. At times it is so severe that morphin is required.

Abdominal distention usually occurs quickly, is frequently severe and is almost without exception in the epigastrium, causing a tumor there, but on account of the distention being due to the enlarged stomach, and the latter occupying an abnormal position, the whole abdomen is sometimes distended. Frequently the outline of the greatly distended stomach can be plainly seen. This abdominal enlargement completely disappears after lavage.

Constipation is the rule. In two of my own cases the first thought was that the symptoms were due to intestinal obstruction. Occasionally, however, diarrhea occurs.

Collapse.—The patient is frequently almost totally collapsed. The face is pinched and anxious. The eyes are sunken, the pulse is running, the breathing is rapid, the patient gives every evidence of immediate dissolution.

Splashing sounds.—By placing one hand upon the lower portion of the abdomen, and making a quick percussion of the portion occupied by the tumor of the abdomen, usually a splashing sound can be detected, which is characteristic of dilatation of the stomach.

Peristaltic movements of the stomach area can occasionally be seen. This has been noted in only a few instances and is apparently not as marked in cases of acute dilatation as it is in cases of chronic dilatation.

Conditions to be Differentiated from Acute Dilatation of the Stomach

General intestinal distention not due to obstruction or peritonitis
Peritonitis due to perforation or to extension of inflammation
Intestinal obstruction
Pancreatic cyst
Uremia
Acute hemorrhagic pancreatitis
Inflammation of the gall-bladder
Dilatation of the colon
Postanesthesia vomiting.

General Abdominal Distention.

General abdominal distention is common in many acute infections, particularly in typhoid fever and in pneumonia, and is frequently accompanied by gastric dilatation. In simple distention, peristalsis may be heard over the entire abdomen. The outline of the stomach cannot be seen, and distention will be very little altered by lavage. There is no vomiting; often there is diarrhea. Frequently in these cases a rectal tube will relieve the tympany.

General Peritonitis.

Here there is the same rapid distention as in gastric dilatation but the outline of the stomach cannot be seen. There is much more pain and tenderness than in dilatation, and there is no splash, but the same collapsed condition of the patient is evident. Above all, the stomach tube does not dissipate the distention. There is usually fever and leukocytosis.

Intestinal Obstruction.

Three cases which occurred in pneumonia, and which came under my supervision, were believed to be due to intestinal obstruction—indeed the picture was very much like it: abdominal pain, vomiting (in two instances almost fecal in character), great distention and constipation. The later theory that there is always an obstruction where the mesentery crosses the duodenum, explains the likeness of the pictures. In intestinal obstruction, however, the distention is general over the entire abdomen, whereas in acute dilatation of the stomach the distention is likely to be in the epigastrium, or at least greater in that position. Sometimes a marked epigastric tumor is seen, occupying the entire epigastrium on the left hypochondrium and also the lower epigastric region, and in rarer instances the outline of the distended abdomen can be seen through the abdominal wall. Careful passage of the stomach tube will cause the immediate disappearance of the abdominal distention in gastric dilatation, and will not do so in general abdominal distention.

Pancreatic Cyst.

Dilatation of the stomach has been mistaken for this condition, but in cyst there is evidence of a true mass; this mass is dull to percussion; the stomach tube will not cause its disappearance; there is no collapse in the case of pancreatic cyst.

Uremia.

The dull, unconscious condition of the patient in uremia is not like the rather active delirium of acute dilatation of the stomach. There is no collapse, nor is there distention. Examination of the urine will make the diagnosis positive.

Acute Hemorrhagic Pancreatitis.

Acute hemorrhagic pancreatitis has the same sudden onset with collapse, but here the distention is local and not confined to the stomach. It is easily differentiated by means of the stomach tube. It occurs without any exciting cause such as operation, acute infection or overeating.

Infammation of the Gall-bladder.

Cholecystitis has as its characteristic symptoms, pain, fever and local tenderness, often with leukocytosis. The stomach is not dilated.

Dilatation of the Colon.

Dilatation of the colon is not acute. The distention is apt to be more general; it cannot be relieved by a stomach tube, but the patient may at least gain temporary comfort through the use of a rectal tube.

Vomiting Due to Anesthesia.

This conditon is a common one. There is no collapse. If there is abdominal distention, it is not due to dilatation of the stomach; it usually occurs immediately after the operaton. It is of great mportance that an early differential diagnosis should be made by use of the stomach tube. Gastric dilatation when allowed to continue, may easily be fatal.

5. Peptic Ulcer

(*Duodenal Ulcer—Gastric Ulcer*)

The truth of the following statement, written by William J. Mayo, in the St. Mary's Hospital collection of papers for 1911, cannot be better illustrated than in the diagnosis of gastric and duodenal ulcers from each other and from conditions resembling them: "Until surgery came to our aid we had no means of directly inspecting the abdominal contents during life, and this led to too great dependence upon clinical data derived from indirect methods."

Surgeons, among whom the Mayos have been most prominent, have established the facts that "gastric and duodenal ulcers are more often found in the male sex, that chronic duodenal ulcer is more common than gastric, and that in the large majority of cases the ulcer is single"—this notwithstanding the fact that for years the opposite was supposed to be the fact.

Symptoms.—Pain is among the symptoms common to both gastric and duodenal ulcer; it usually occurs one to three hours after eating when the ulcer is in the pyloric region, whether duodenal or gastric. When the ulcer is nearer the cardiac end of the stomach, the pain occurs earlier, following the ingestion of food. The pain of ulcers in the pyloric region is relieved by taking food or an alkali; the attacks often occur in the night and come on in paroxysms. Mayo says: "Hunger pain and food relief are very typical of ulcer." This is true, yet in our histories we often find such a report but no ulcer is found; though usually where no ulcer is found the history is not as sharp cut as in ulcer cases.

Hyperchlorhydria and hypersecretion are the rule in the early stages of both forms of ulcer.

Hemorrhage, blood being either vomited or passed by the bowel, occurs frequently, but often one must employ leading questions to obtain a statement of facts from the patient. In later stages there is marked retention of food when the ulcer is in the duodenum or in the pyloric end of the stomach. The obstruction upon which the Mayos depend most is the retention of finer articles of food, ten to twelve hours after ingestion.

The stomach contents—an extract from Graham's and Guthrie's tables published in 1909, is of importance and is here given:—

ACIDITY IN

Ulcer of Stomach and Duodenum..	250 cases	Free HCl	Present in 237 cases
Carcinoma	150 "	" "	" " 70 "
Pyloric Spasm	100 "	" "	" " 84 "
Functional Neurosis	100 "	" "	" " 95 "

It will be seen that free HCl is more common in functional neurosis and pylorospasm due to appendicitis and gall-stones, and it is this condition which so often causes trouble in diagnosis.

Important symptoms in making a diagnosis are the periodicity, chronicity, and character of the pain. The attacks of pain cover several days or weeks at a time, with intervals of apparently good health; these intervals may last weeks or months. The pain is more apt to occur in the fall and spring; the cases often last years, many painful attacks having occurred.

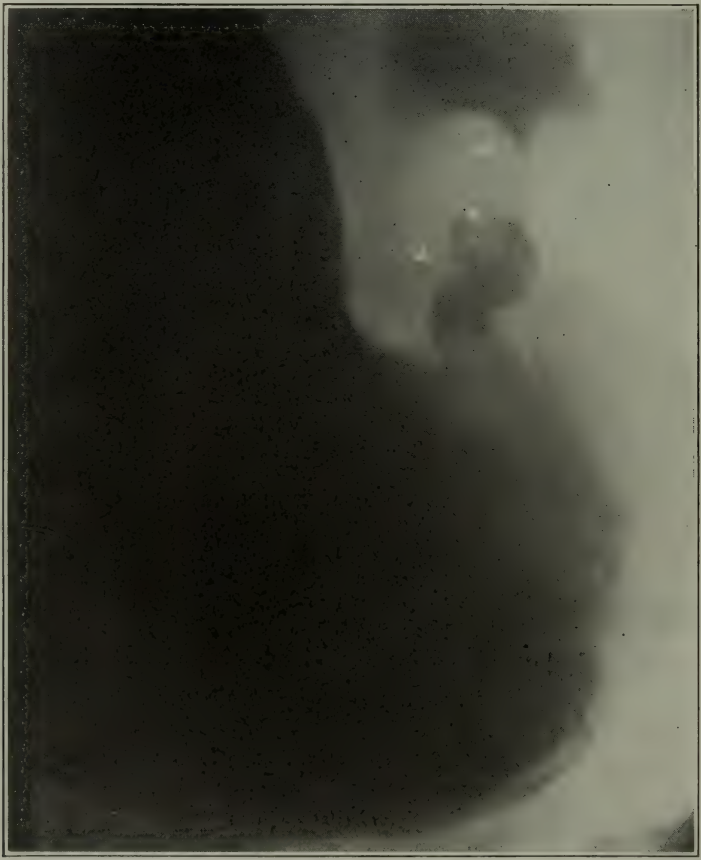


Fig. 52.—X-ray Picture of Ulcer of Stomach. The Arrows Show the Indentations. H. K. Pancoast, fecit. (Original Observation.)

The pain is relieved by eating unless the ulcer be near the cardiac end of the stomach. However, failure to obtain relief is so rare that *relief of pain by taking food or an alkali is a cardinal symptom of peptic ulcer.*

Hemorrhage from the stomach or bowels, either macroscopic or occult, is an important symptom, but compilation of statistics shows the condition is not nearly so important from a diagnostic standpoint as supposed.

Food retention is important and confirmatory of other symptoms. However, it is the cases without retention and proven ulcer, which give much difficulty to the diagnosis.

The x-ray often furnishes valuable information by showing dilated stomach, deformed stomach, and the presence of adhesions. In serial pictures, when rapidly taken, after a bismuth meal, the presence of a defect in the duodenum or the pyloric end of the stomach will often make the diagnosis certain (See Fig. 52).

Conditions to be Differentiated from Gastric Ulcer

Appendicitis
Diseases of gall-bladder and ducts
Gastric neuroses
Gastritis
Gastric crises
Carcinoma.

All these are often confused with gastric ulcer, as spoken of in another chapter. The word dyspepsia has since time immemorial caused a "multitude of sins," and it is only since surgery has enabled us to check off our errors, that we are beginning to learn. Even yet while we may be in full possession of knowledge of the danger of mistakes, errors still occur, not from wrong interpretation but from our unwillingness to believe *that simple dyspepsia often spells disaster.*

APPENDICITIS.

Appendicitis may give rise to pain in the epigastrium, and often it is first felt in that region, but usually the attacks are localized in the appendiceal region. There is tenderness and resistance in that vicinity. In acute cases there is fever and leukocytosis; sometimes a tumor can be felt in that region. The attacks of pain do not have the same periodicity, nor is the pain relieved by food, as in peptic ulcer.

DISEASES OF THE BILE PASSAGES AND GALL-STONES.

These conditions are characterized by pain in the epigastrium; it is usually sudden and not relieved by eating and is perhaps more severe than the pain of gastric ulcer. Often the patient becomes collapsed. Vomiting does not give the same relief as in ulcer, and almost always there is need of morphin for relief.

Hemorrhage rarely occurs in gall-stones. Jaundice may occur after gall-stones, but it must be remembered that it is not a constant accompaniment. There is seldom loss of weight. The characteristic hunger pain relieved by eating or taking an alkali and the periodicity and chronicity are not marked features. Often a differentiation is not possible before operation.

GASTRIC NEUROSES—GASTRITIS.

Gastric neuroses and gastritis are constantly diagnosed when ulcer should be the decision. The lack of relief by eating, dependence upon the kind of food taken, relief by lavage, change of surroundings, absence of hemorrhage or loss of weight, are valuable diagnostic symptoms. Occasionally persons suffering supposedly from these conditions lose much weight because of a prescribed diet, amounting to starvation. This must be carefully excluded.

GASTRIC CRISES.

Gastric crises need only to be remembered to be diagnosed. There are the unquestionable signs of changes in the nervous system which are not present in ulcer.

CARCINOMA.

Carcinoma, often the end result of peptic ulcer, may be thought present because of a tumor which is really due to fibroid thickening around an ulcer, but the diagnosis can be substantiated by the greater lack of acid stomach contents, more frequently blood, and by the x-ray picture. (See Carcinoma of Stomach.)

6. Cirrhosis ventriculi

(*Plastic Linitis*)

Cirrhosis ventriculi is a rare condition of sclerosis of the walls of the stomach characterized by constant vomiting, emaciation, and a tumor having the feel of a cylindrical mass and without the gradual appearance of a tumor often felt across the whole epigastrium.

It is constantly mistaken for other forms of gastritis.

Constant vomiting of even small quantities of food with an oblong tumor in the stomach, will help to make the diagnosis. In a case which came under the writer's observation the wall was found to be the seat of scirrhous cancer.

The absence of tumor will distinguish it from various other forms of gastritis.

The prolonged life and the lack of metastasis, will distinguish the non-malignant from the malignant form, but operation should be undertaken in the presence of such symptoms and a mass, not only to establish a diagnosis, but for curative purposes. Riesman has reported such a case.

7. Cancer of the Stomach

It is with some hesitancy that the author gives rules for the diagnosis of gastric cancer. These are positive when they exist, but when one waits for their appearance in order to make a positive diagnosis of cancer of the stomach, he frequently waits until there is a tumor, and the appearance of the latter signifies that the method of relief—surgical interference—is usually past its time of efficiency. After a case has run the gamut of rational treatment and exhaustive diagnosis without relief, it is much better to err on the side of safety and operate than dally too long with so-called treatment.

Symptoms.—The symptoms of gastric cancer are eructation of gas, vomiting, pain, loss of weight, hematemesis and tumor. As has been said in the chapter on gastric ulcer, the symptoms frequently follow those of gastric ulcer and imperceptibly drift into those of cancer. The pain becomes more constant; the intermissions common in ulcer are not so frequent and are less prolonged; the effect of taking food is less marked in its relief; emaciation appears, and the stomach contents vary somewhat. The pain of gastric ulcer is merged into a constant gastric distress often made worse by eating.

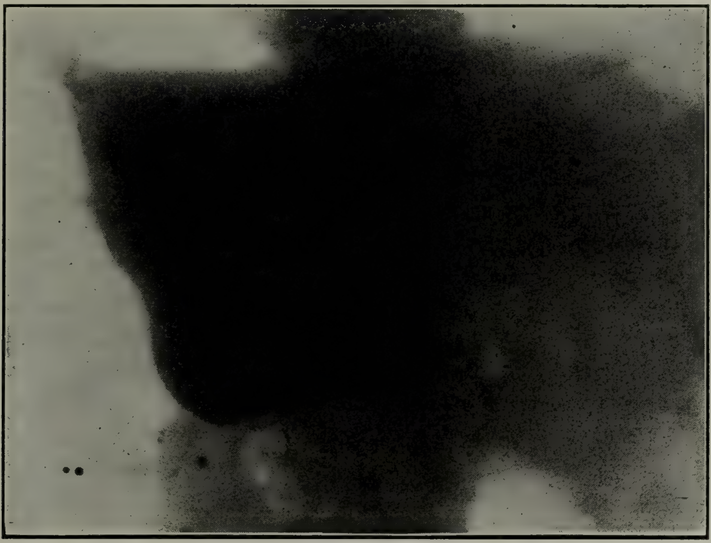


Fig. 53.—Radiogram of Carcinoma of Stomach, Showing Lack of Shadow at Pylorus. (Original Observation.)

Of 150 cases at Mayo's clinic, in the gastric contents from those suffering from cancer of the stomach, free hydrochloric acid was present in 70 cases and was absent in 80 cases, showing a decided proportion of anacidity as compared with those of gastric ulcer. Blood was present in 80 cases, lactic acid in 64 cases, food remnants were present in 63 cases. Oppler-Boas bacilli are frequently present. Loss of weight and lack of appetite are constant in gastric cancer, and are always suspicious symptoms when they occur with the preceding history of gastric ulcer.

The tumor makes the diagnosis positive when connected with the above symptoms. Inflation of the stomach through a stomach tube will frequently, though not always, show a dilated stomach. Examination by the x-ray shows an interference of peristalsis and sometimes a notching of the stomach wall (Fig. 53).

Conditions to be Differentiated from Cancer of the Stomach

This condition must be distinguished from:

Ulcer
Gall-bladder disease
Appendicitis
Pernicious anemia
Tuberculosis
Gastric neuroses
Chronic dilatation of the stomach.

The important point in differentiation is that *a diagnosis be not long delayed:* better to operate for a case of supposed gastric cancer and find only one of the other conditions mentioned than to delay the diagnosis until a tumor appears, *for the appearance of a tumor,* as I have said, *usually makes an operation valueless.*

ULCER.

In ulcer of the stomach or duodenum the pain usually comes on two or three hours after ingestion and is relieved by eating. There is retention and hyperacidity. A tumor may exist and retention occur simulating the tumor and retention of gastric carcinoma. *Only an operation will make the diagnosis positive.*

GALL-BLADDER DISEASE—APPENDICITIS.

Gall-stones and chronic appendicitis are characterized by indigestion and pain and often loss of weight; there are no Oppler-Boas bacilli; the pain is usually in the gall-bladder region or in the region of the appendix.

PERNICIOUS ANEMIA.

Pernicious anemia often simulates carcinoma of the stomach. The blood picture of pernicious anemia, the history of marked amelioration of the symptoms when the patient seems to have recovered—from which he soon relapses and during the relapse shows blood impairment, the absence of tumor and the fact that the anemia is much more extreme than it is in carcinoma, all speak for pernicious anemia. Emaciation is much less marked. The x-ray does not show any change in the stomach.

TUBERCULOSIS.

Tuberculosis might be mistaken for carcinoma of stomach because of emaciation and vomiting, but care in physical examination of the lungs, the presence of tubercle bacilli and of cough will help to determine the diagnosis.

8. Hypertrophic Stenosis of the Pylorus

Symptoms.—Hypertrophic stenosis of the pylorus is indicated by emaciation, vomiting of food, a mass in the region of the pylorus, and gastric peristalsis.

The *congenital condition* may be preceded by hyperacidity and expulsive vomiting before any mass can be felt.

Vomiting from other causes in children must be differentiated by the absence of a pyloric mass, gastric dilatation and peristalsis, and the presence of causative factors, such as improper food.

Differentiation.—When the case occurs during *adult life,* it must be differentiated from:

Carcinoma
Occlusion as the result of gastric ulcer
Fecal mass.

These conditions must be differentiated by the history of the cases and examination of the stomach contents. *Operation will alone be the only safe diagnostic means.*

The constant presence of an operable mass calls for operating as a diagnostic measure; fecal mass can be made to disappear by the use of purgatives. Purgatives do not affect a mass due to organic conditions.

9. Hematemesis

Etiology.—Vomiting of blood may arise from several different causes: hemorrhage from the *nose* when the blood has been swallowed; hemorrhage from the *lungs* when some blood has been swallowed and then vomited; hemorrhage from the *pharynx*—a rare cause; hemorrhage from the *esophagus* due to traumatism caused by swallowing an object too large for the caliber of the esophagus, causing *laceration* and consequent bleeding; hemorrhage due to *rupture of a vein* at the cardiac end of the esophagus where a varix has formed; hemorrhage from *the stomach due to cancer or ulcer; injury to the stomach caused by swallowing foreign matter,* or an actual extensive traumatism; a hemorrhagic condition of the *gastric mucous membrane* when the tissue bleeds at any point but where there is no real abrasion of the mucous membrane; *cardiac disease,* especially mitral stenosis; *hemophilia; toxic conditions* (Osler says many cases follow operation for appendicitis); *rupture of an aneurism into the esophagus or stomach.*

The *most important* in point of differentiation of these is perhaps bleeding from esophageal varices as contrasted with bleeding from an ulcer of the stomach.

In newborn infants, vomiting of blood may occur as one of the symptoms of a dyscrasia which gives rise to melena and bleeding from any of the mucous membranes—vicarious hemorrhage.

The mere statement of an attendant can seldom be taken as an accurate history as to actual blood vomited; often the dark changed gastric contents is considered by an attendant as blood when none is present. On the other hand partially digested blood may not be recognized. In doubtful cases the only safe rule to follow is to have the material examined either by means of a chemical test or the microscope.

Diagnosis.—There is frequently some difficulty in deciding whether blood has been vomited or coughed up. This may usually be settled by attention to the following points:

HEMATEMESIS (*Blood vomited*)	HEMOPTYSIS (*Blood coughed up*)
Blood dark red, or dark and granular	Blood bright red at first, mixed with sputum
Mixed with food particles	No food
Acid in reaction	
Frequently clotted	Usually frothy, sometimes small clots
History of nosebleed	History of cough preceding and following
Gastric disturbance	
Heart disturbance	Signs of consolidation of the lungs
Tarry stools	No tarry stools

As to the actual source of the vomited blood:

Epistaxis and bleeding from the pharynx can be diagnosed by careful examination of the nose and pharynx. The blood can be seen either at the nares in epistaxis or in the pharynx where there is an ulcer or bleeding vein.

Bleeding due to traumatism of the esophagus can be discovered by the history of passage of a tube or by swallowing some rough article.

Esophageal bleeding from varices can be suspected where there is a history of splenic or hepatic disease and where the patient is an alcoholic. There is no distinct history of gastric pain preceding the hemorrhage. The bleeding from these varices, when they are due to portal obstruction or cirrhosis, is usually sudden, often in large amounts and without the least history which points toward a previous gastric disturbance. If the varices are the result of stasis due to cardiac insufficiency, the symptoms and physical signs of that condition will be present.

In *cancer of the stomach* and in *ulcer of the stomach,* there is frequently both a previous and succeeding history of gastric disease, with sometimes tarry stools, and frequently occult blood in the stool.

Bleeding due to cardiac disease may be suspected when there is present an actual cardiac lesion, especially when the lesion is one of mitral stenosis, and where there is no history or physical examination to substantiate any one of the other causes of blood vomiting.

Hemophilia, or a purpuric tendency, as a cause of hematemesis may be accounted for by the tendency to bleeding from an abraded surface, by the history of bleeding in males of the family, and by the history and presence of hemorrhages in other portions of the body.

A septic condition as a cause of the bleeding may surely be discovered by fever, leukocytosis, and other symptoms of that condition.

Vicarious hemorrhage, occurring at the expected time of a delayed menstrual period, perhaps does occur, but the time is long past when one can with reason use the term "vicarious" for a gastric hemorrhage; it is certainly extremely rare. Every possible diagnostic means should be used before "vicarious" is allowed to stand as a diagnosis. It is much like the term "indigestion," often used to cover a number of diagnostic faults. Perhaps in the very few cases reported from a reliable source where the bleeding has been repeated, there may be reason for the diagnosis, but isolated hemorrhages should rarely be so diagnosed. The writer in a rather large experience has never seen a case in which the diagnosis was justified.

10. Pylorospasm

Etiology.—This is a sudden, severe, spasmodic stricture of the pylorus, causing pain, epigastric distress and often vomiting. It is frequently due to the presence of hyperacidity, and often to gall-bladder disease, appendicitis or gastric ulcer. The presence of a pylorospasm may not mean an actual organic disease, but certainly when there is a presence of a pyloric tumor which can be intermittently felt, one is much safer in believing the condition to have an organic basis.

Diagnosis.—Pylorospasm can be differentiated from organic conditions by persistence of the condition, by the fact that a tumor cannot be felt, and by the use of a thread, which when swallowed will have less tendency to enter the duodenum if the case is organic in character.

Examination by the x-ray, firstly by the use of the fluoroscope, and secondly by the taking of serial pictures, will show deep peristaltic waves. These waves due to peristalsis can be distinguished from faults due to contraction in the wall, the result of ulcer or carcinoma, by the fact that they are seen to affect different portions of the wall both in the fluoroscopic view and in the plate, while faults due to ulcer are exactly in the same place in all the plates.

If the condition persists, however, in spite of treatment, the best diagnostic means is an exploratory incision. The condition often occurs in newborn children; here it can be diagnosticated by otherwise causeless vomiting and the occasional appearance of a mass in the region of the pylorus.

11. Neuroses of the Stomach

Under this caption are included the various and varied conditions popularly known as "dyspepsia," which have no basis of organic change either in the stomach itself or in the adjacent organs, such as the pancreas, gall-ducts and gall-bladder, or in the vermiform appendix. In making a diagnosis of one of the neuroses, the most vital point is the exclusion of the above mentioned conditions, for very many cases which are "dyspepsia" in the minds of the laity—and unfortunately, too, in the minds of some physicians—are really appendicitis, cholelithiasis or its results, pancreatitis or gastric ulcer or cancer. Indeed, the writer feels quite certain that the sum total of good would be greater if we recognized all of the neuroses as having a foundation in some organic change. This would be an error, of course, but we then would not be subject to the biting chagrin of suddenly being face to face with a case of general peritonitis, the result of a neglected appendicitis or gastric ulcer, or when a long delayed operation has been performed, we would have the unpleasant duty of announcing to the patient and family that the fear, danger and expense coincident with an operation have been practically useless because of the presence of an advanced carcinoma.

As has been insisted several times in this volume, every possible means of diagnosis, a rational method of treatment, including, of course, proper diet and work, must be continuously employed in all cases of dyspepsia. If in the course of a few weeks of treatment our methods of diagnosis have given us no real and positive knowledge that we are *not* dealing with an organic condition, then an exploratory operation should be made to decide the point.

(*a*) ***Hypermobility***

Hypermobility of the stomach is the condition in which the viscus empties itself too quickly, either as the result of a hypersecretion or of hyperacid secretion. It can be diagnosed by a test meal and finding the stomach entirely empty at the end of one hour. Care must be taken that the apparent emptiness of the stomach is not in reality due to some fault either in the passage of the tube or in the attempted removal of the contents. By inflating the stomach one can be sure that it is of normal size and not one of the curiously contracted gastric cirrhoses which occasionally occur.

Examination with a fluoroscope will give most valuable information; a bismuth meal is given and the rate of the movement can be seen by means of the fluoroscope. It must be taken into consideration, that here, too, we are gradually becoming convinced that hypermobility is one of the conditions often dependent upon gastric ulcer or some other organic change.

(*b*) ***Peristaltic Unrest***

This is a condition where the peristalsis is so sudden and violent that not only is the patient aware of the movements of the stomach, but a rumbling noise can also be distinctly heard by the bystander.

The condition is of course diagnosed by the presence of these symptoms, or really by the persistence and violence of the movements and resulting sounds. It is not to be confused with borborygmus, which is the result of noisy movements of the intestines due to indigestion from many causes.

(*c*) ***Aërophagia***

Aërophagia is the result of the individual sucking air into the stomach exactly as a horse aspirates in the process of "cribbing." It results in noisy eructations continued over a long period of time, occurring usually in hysterical individuals; it also occurs after an attack of angina pectoris such as described in the article dealing with that subject, the attack simulating an attack of indigestion.

It is extremely difficult to detect the individual in the act of swallowing air, but occasionally the stomach can be suddenly seen to distend just before the eructation. If the patient be prevented from closing the mouth by the use of a gag or by putting a cork between the teeth, the eructations will cease, as the air cannot be sucked into the stomach. This must be distinguished from belching of gases formed in the stomach.

The patient is usually free from any of the ordinary signs of indigestion; he may be hysterical; or it may be due to organic heart disease above described.

(*d*) ***Nervous Vomiting***

Nervous vomiting may occur in adults who are hysterical, or in children. The vomiting, especially in children, may occur during the act of taking food; the food is returned entirely undigested; there is no sensation of nausea, and the character of the food is unchanged. The character of the food does not influence the attack. This form of vomiting sometimes occurs when an individual is subjected to the sight of blood, or some similar unpleasant experience.

Conditions to be Differentiated from Nervous Vomiting

This condition can be mistaken for vomiting due to:

Acute indigestion
Dilatation of the stomach
Acute infections
Vomiting of organic nervous conditions.

VOMITING DUE TO ACUTE INDIGESTION.

Acute indigestion is the result of improper food, proper food swallowed too quickly, undue exercise or excitement after ingestion. The nervous vomiting comes on while eating—independently of the kind of food. There is usually nausea and pain with acute indigestion. It lasts longer than nervous vomiting.

VOMITING DUE TO DILATATION OF THE STOMACH.

Dilatation of the stomach gives rise to vomiting, sometimes without nausea; the quantity of vomitus is large and is often ill-smelling and fermented. The dilatation can be detected by the proper examinations, x-rays and stomach tube.

VOMITING DUE TO THE ACUTE INFECTIONS.

This may begin with sudden painless vomiting, but fever, the result of infection, soon follows, together with the specific signs of the disease.

VOMITING OF CEREBELLAR DISEASE AND LOCOMOTOR ATAXIA.

These can be distinguished by the essential signs of those conditions.

CARDIOSPASM.

See article.

(*e*) ***Gastralgia***

Intermittent pain in the epigastrium simulating that of gastric ulcer, is probably a true entity. The pain comes on sometimes after eating;

there is no blood in the stools or in the vomitus; there is likely to be anacidity.

Differentiation.—Greatest care must be taken to distinguish this condition from GASTRIC ULCER. The fact that the condition is amenable to treatment, the absence of blood in the stools and vomitus, the less likelihood of positive hyperacidity—all these make the case one of probable gastralgia. Repeated attacks, however, without discoverable cause, should lead to surgical interference for diagnosis and relief.

(*f*) *Rumination*

Rumination occurs in nervous individuals and sometimes in those with atony of the stomach. It may become involuntary (Riesman). It is the act of regurgitating food into the mouth and rechewing it. It is purely neurotic. There is no complaint of the symptoms on the part of the patient.

About the only condition with which it can be confused is regurgitation of food from esophageal stricture or cardiospasm, but in neither of these conditions is the food rechewed; the patient complains of inability to swallow. A stomach tube or a sound will at once decide the presence of a stricture or spasm.

(*g*) *Cardiospasm*

This is a spasmodic closure of the cardiac end of the stomach, characterized by pain beneath the lower end of the sternum as though the food lodged there; later, by regurgitation of the food in the form in which it was swallowed, and finally by a uniform dilatation of the esophagus (Fig. 54).

The diagnosis is suggested by the rather sudden inability of a nervous individual to swallow, accompanied by pain at the lower end of the esophagus, and later by the regurgitation of food.

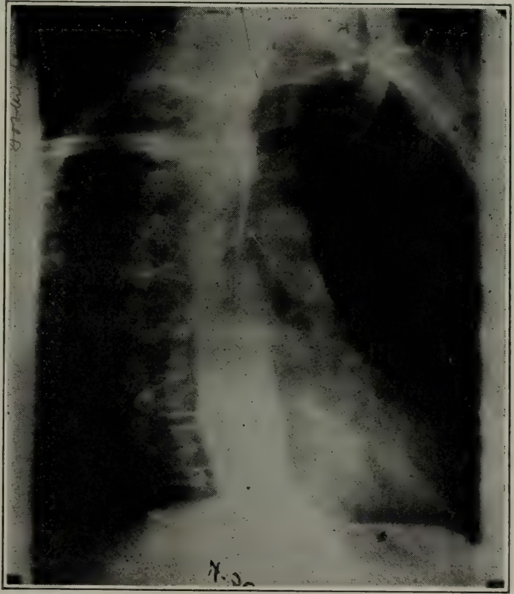


Fig. 54.—Dilatation of Esophagus Due to Cardiospasm. H. K. Pancoast, fecit. (Original Observation.)

Diagnosis.—This can be made by the passage, firstly of a stomach tube which will usually be retained at the cardiac end of the stomach. The passage of a solid bulbous bougie, however, can be accomplished without trouble. If an organic stricture exist both will be retained. The administration of a bismuth meal and the taking of an x-ray picture will show the bismuth held above the cardia, and if the esophagus be dilated, that will be shown in the radiograph. Frequently a solid bougie may be retained at the lower end of the esophagus even though the stricture is spasmodic. This is due to the fact that a dilatation has occurred in the esophagus causing a sacculation at the lower end. The bougie may become caught in this sacculation and refuse to enter the stomach; this difficulty can be easily overcome by using a Plummer sound (Fig. 55) which can be threaded. The patient is given six yards of ordinary silk. He swallows three yards of this in the evening, the other three yards the following morning, retaining the end at the teeth so it will not go into the mouth. The proper sized bulb is threaded over this silk and passed. If the stricture is not organic the thread will guide the bulb into the stomach without any trouble; this does away with any danger of perforation, for the silk directs the point of the bougie in the proper channel. Of course, if the silk has not entered the stomach and intestine it will be impossible to use it because when traction is made on the end at the teeth it will be discovered that it is loose at the lower end when traction is attempted. It will not be fastened as it would be if it were carried into the intestine. Spasm of the esophagus itself occurs in the course of the esophagus and not at the extreme lower end. An x-ray picture will show retention above the point of spasm.

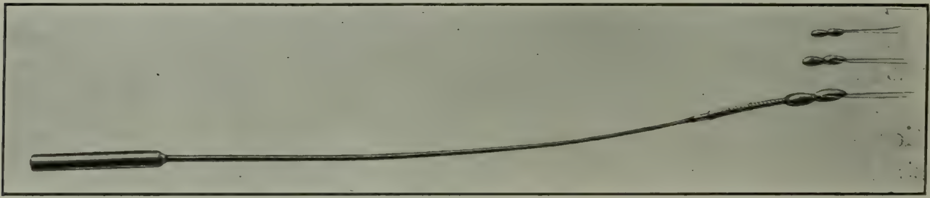


Fig 55.—Plummer's Sounds. Tip Threaded.

Treatment by dilatation by means of a rubber dilatable bag will cure the condition. An organic stricture cannot be so cured.

Conditions to be Differentiated from Cardiospasm

It must be distinguished from:

Spasm of the esophagus

Stricture of the esophagus due either to neoplasm or pressure on the esophagus

Diverticulum of esophagus.

STRICTURE OF THE ESOPHAGUS.

An organic stricture is differentiated by rapid emaciation of the patient, the inability to pass any sort of sound, and finally by repeated x-ray pictures showing the constant stricture.

DIVERTICULUM OF THE ESOPHAGUS.

A diverticulum can be shown to exist by the fact that sometimes a bougie will be caught at a certain point, and at other times it will pass readily. Here the Plummer sound may also be used; finally an x-ray will demonstrate the shadow of a sacculation.

12. Hyperchlorhydria—Hyperacidity—Superacidity

This condition is often a symptom of diseased conditions of the stomach, or it may be a simple gastric neurosis. There is complaint of burning pain and "heartburn" when the stomach is empty—this pain often relieved by eating and of the eructation of sour material. The pain may radiate to the back. Examination of the stomach contents will show the hydrochloric acid content to be above or at the upper limit of normal in some cases, often reaching as high as 40 or 60.

The larval form of hyperacidity is that condition with symptoms of hyperchlorhydria without actual increase in the hydrochloric acid of the stomach contents. The finding of excess of hydrochloric acid in the stomach contents, of course, makes the diagnosis of hyperchlorhydria complete, but one must not be satisfied with that diagnosis alone—it is dangerous.

Conditions to be Differentiated from Hyperacidity

The symptoms of gastric ulcer, gall-bladder disease and appendicitis so frequently exactly simulate the simple neuroses, as has already been stated, that the very elect may occasionally be deceived.

GASTRIC ULCER.

Gastric ulcer is indicated by the chronicity and periodicity of the attacks. The pain in typical cases is relieved by taking food and by the use of an alkali; there is almost without exception retention of the stomach contents as shown by the x-ray; there is also likely to be occult blood in the gastric contents or stool.

GALL-BLADDER DISEASE.

Gall-bladder disease while it comes on in periodical attacks is not so regular as those of gastric ulcer. The pain is not so much relieved by food as is that of gastralgia and there is apt to be tenderness on local examination, while there may be no jaundice.

APPENDICITIS.

Appendicitis is likely to be less affected by food. There is usually a spot of tenderness in the right iliac fossa, and there may be leukocytosis.

However, gastralgia, gastric ulcer, gall-bladder disease and appendicitis are often entirely inseparable one from another.

When every effort has been made to make a diagnosis, and careful dietetic and medical treatment has failed to cure the patient, certainly an exploratory operation is not only warranted but demanded. As was stated in the beginning of this chapter, it is much better in these days of certain surgery, to have done what seems to be an unnecessary surgical procedure, than to allow a patient to die of one of these conditions which can be remedied. This means, of course, that every reasonable diagnostic procedure be used and that a skilled surgeon, one who has had preliminary and much practical experience, be taken as consultant. No one can be more opposed to useless surgical procedures than the writer, but *when one has reached the limit of his diagnostic skill and the patient still suffers, operation is not only indicated but imperative.*

13. Supersecretion

(Reichman's Disease)

This is a form of indigestion characterized by continuous or intermittent secretion of highly acid gastric juice. There is pain, much gastric juice—highly acid; there may be vomiting, and sometimes dilatation of the stomach.

The differential diagnosis here is much the same as holds in the diagnosis of simple hyperacidity; exactly the same precautions against the same errors must be taken.

G. Diseases of the Intestines

1. Diarrhea of Children

This condition can be mistaken for little else. The symptoms vary from simple frequent stools to a large watery evacuation, resembling the stools of cholera, with high fever and accompanied by rapid pulse and much emaciation.

Diagnosis.—In time of cholera epidemics this latter form might be mistaken for cholera, but the stool examination will easily make the diagnosis positive. Cholera vibrios will be seen in those suffering from Asiatic cholera and will be absent in diarrhea due to simple enteritis. In summer time when all diarrheas of children are most common, the condition might be mistaken for the early diarrhea of typhoid fever, and, as typhoid fever is relatively uncommon in children and as it is notoriously difficult of diagnosis, all suspicious cases with fever must have the blood examined for Widal reaction or must at once have a blood culture made. If the case is one of typhoid fever there will be a continuation of the fever, and the spleen will enlarge.

Investigation of Food.—The diarrhea of children calls at once for investigation of the food supply—bad milk, unripe food, and gormandizing are all prolific causes of diarrhea.

2. Catarrhal Enteritis

Etiology.—This is a mild inflammatory condition of the intestine, due to some irritation of the mucous membrane. Frequently this irritation is the result of overeating, of taking improper food or of improperly cooked food. Occasionally the attack is due to perfectly proper food, taken and cooked properly, but followed or preceded by undue mental excitement or physical work. It may also be due to the formation of certain toxic substances.

Symptoms.—The symptoms vary with the character of the causative agent and the degree of neglect of conditions. Diarrhea is the constant symptom—at first a normal stool soon followed by a liquid stool, with more or less tenesmus. The stools may be large and watery after the first onset, followed by frequent small stools containing blood and mucus and accompanied by more or less tenesmus. The degree of tenesmus and the amount of blood and mucus indicate the degree of involvement of the colon, ileocolitis or colitis alone being always indicated when these appear.

Conditions to be Differentiated from Catarrhal Enteritis

In differentiating this condition, due as it frequently is to improper food or to the manner of taking food, one must remember that it is really only a symptom.

APPENDICITIS.

Appendicitis which is usually accompanied by constipation, has occasionally for one of its initial symptoms diarrhea or dysenteric attacks. In this condition, however, there is fever, localized pain and tenderness with leukocytosis. The mistake is more likely to occur when there is vomiting of undigested food, because one is more apt to attribute the entire attack to the ingestion of improper food.

TYPHOID FEVER.

The diarrhea may be the result of some such systematic disease as typhoid fever. Prolonged diarrhea, especially when no cause can be discovered and when accompanied by fever, at once calls for a careful examination for the symptoms and signs of typhoid fever, as pointed out in that chapter.

The method of diagnosis here must be by extremely careful history and physical examination, and especially by the use of the temperature chart and the blood culture as well as the blood count; one must make sure of the absence of any septic condition.

Typhoid fever may begin with a severe diarrhea. Here, the history, the characteristic temperature curve, the absence of a leukocytosis and the presence of a Widal reaction will make the diagnosis of typhoid positive.

Conditions to be Differentiated from Severe Simple Enterocolitis

A severe, simple enterocolitis, *especially where the colon is particularly involved*, may be mistaken for:

DYSENTERY, EITHER OF THE BACILLARY TYPE OR THE AMEBIC TYPE.

Here the presence of dysentery bacilli or amebae in the stools will make an absolute diagnosis. The symptoms, however, of the two conditions, enteritis and dysentery, differ in certain particulars. In dysentery there are, as a rule, much more violent symptoms. There is evidently a toxic state, as indicated by the more severe systemic conditions present, shown by emaciation, by fever and, at times, by nephritis.

CHOLERA.

The severe acute attacks of enteritis, known frequently as cholera morbus or cholera nostra and which are characterized by many of the symptoms of Asiatic cholera—vomiting, severe abdominal pain, large choleraic stools, rapid emaciation due to loss of water from the tissues, and cramps in the muscles, especially in the muscles of the calves—may be very readily mistaken for epidemic cholera. The diagnosis is particularly important at the time of epidemics of cholera. At such times a bacteriological examination of the stools of all suspicious cases should be made. True cholera will show the presence of comma bacilli, which will be entirely wanting in the simple enteritis.

TUBERCULOSIS OF THE INTESTINE.

Tuberculosis of the intestine may simulate a simple diarrhea, but the chronicity of the case, together with signs and symptoms of tuberculosis in other portions of the body, will help to make the diagnosis.

ULCERATIVE PHLEGMONOUS DIPHTHERITIC ENTERITIS.

Ulcerative phlegmonous or diphtheritic enteritis can scarcely be mistaken for any other condition: there is much depression; it is usually secondary to some other condition.

ULCERATION OF THE COLON.

Ulceration of the colon, which gives rise to frequent stools with much tenesmus, may be simulated by any inflammatory condition—such as carcinoma, etc.—of the lower bowel, or even by inflammation of the pelvic organs or a tumor in that position. One of the writer's cases of dermoid cyst of the ovary had as the first symptom typical dysenteric stools containing blood and mucus; this was due to pelvic pressure. Digital examination is of great importance.

3. Appendicitis

Symptoms.—Appendicitis, acute or chronic inflammation of the vermiform appendix, is characterized in the acute stage by abdominal pain severe or slight—frequently most severe in the epigastrium—often accompanied by vomiting.

The vomitus consists of the contents of the stomach, and because it often occurs after a meal, is mistaken for acute indigestion. The pain after a few hours usually becomes localized in the right iliac fossa. In addition to the pain and vomiting, almost without exception there is resistance and tenderness in the right iliac fossa.

The usual spot of greatest tenderness is midway between the anterior superior spine of the ilium and umbilicus—"McBurney's point." Its location may vary somewhat as governed by the position of the appendix. It may be above or below, or it may be even on the left side of the abdomen. Usually accompanying these symptoms and signs, there are fever and leukocytosis. However, a severe attack of appendicitis may occur without much fever and with very little leukocytosis. If the appendix is retrocecal in position, the tenderness in the classical McBurney's point is less marked. If the appendix dips into the pelvis, rectal digital examination may show a tender area or a mass not revealed by examination of the abdomen.

Chronic appendicitis may cause pain in almost any portion of the abdomen, to attacks of recurrent acute appendicitis, and to marked symptoms of indigestion. Almost invariably there is more or less tenderness in the right iliac fossa. Very frequently cases of chronic inflammation are mistaken for simple indigestion.

Diagnosis.—A person presenting himself with the above symptoms must be thoroughly examined in order to establish or rule out the following possibilities:

Appendicitis
Gall-stones, or gall-duct disease
Perforative peritonitis
Gastric or duodenal ulcer
Hyperacidity
Acute gastritis or enteritis
Obstruction from hernia, volvulus, intussusception, constricting bands
Lead colic
Typhoid fever
Pneumonia or pleurisy
Pancreatitis
Acute epididymitis
Carcinoma or abdominal viscera
Tubercular peritonitis
Gastric crisis or tabes dorsalis
Dietl's crisis
Renal colic
Extra-uterine pregnancy
Acute indigestion
Biliousness.

This is a formidable array of possibilities, but they must be considered in detail if we are to escape the opprobrium of considering a dangerous case as a slight illness.

I believe we general practitioners have to answer for more sins in the name of acute indigestion and hyperacidity and of that meaningless term "bilious attack" than for any others in our practice. Hence I put acute indigestion and its congeries last, because all other conditions must certainly be excluded before we have a right to make a diagnosis of indigestion. Of one thing be assured—*if the abdominal pain is accompanied by persistent fever, tenderness and leukocytosis, the condition is certainly not acute indigestion.*

Blood Counts.—Before I proceed, let me digress one moment upon a subject which is of the greatest importance in diagnosis of abdominal conditions—the making of a leukocytic count. It is frequently absolutely necessary that a blood count be made in order to arrive at a differential diagnosis. Hence, every practitioner should be able to make a count himself; if too busy, let him have one made; it is practicable, it is necessary. If every doctor carried with him a blood counting apparatus and used it, I believe it would often avail much more good to the patient than the carrying of a medicine case. It takes but a few minutes to clean a finger, prick it, suck the blood into a pipet and dilute it; the counting can be done at leisure at home. Personally I always have with me a set of blood counting instruments which I have carried for years for my own and my patients' benefit.

Conditions to be Differentiated

ACUTE APPENDICITIS.

Acute appendicitis has the following symptoms—abdominal pain, tenderness, local resistance, vomiting, constipation, frequent painful urination and leukocytosis.

The abdominal pain is extremely acute, sometimes dull; it may at first be general over the abdomen, later, in the right iliac fossa.

Tenderness is present almost without exception and is usually greatest over the McBurney point, midway between the umbilicus and the superior iliac spine. But the *spot of greatest tenderness may be situated high up along the colon, over the gall-bladder region, over the urinary bladder, even on the left side.*

Local resistance is felt almost without exception over the spot of tenderness, usually in the right iliac fossa, but always over the tender area.

Vomiting is a frequent symptom in the beginning of the attack; often undigested food is vomited.

Constipation is the rule, though at times one finds the contrary condition—there may be loose bowel movements dysenteric in type. There is *frequent, painful urination, often with blood and leukocytes* in the urine; this when the appendix dips down toward the bladder.

Leukocytosis is almost the unexceptional rule. Sometimes there is great increase in the cells, and at times it is moderate, but there is usually some increase in the number of white cells.

These are the symptoms typical of appendicitis, but I want to warn my fellow practitioners that, even with these symptoms, they must think of the list of possibilities above mentioned and send their patients to a surgeon for his opinion.

Fever is present as a rule; severe cases may occur with little fever.

GALL-STONES.

Gall-stones may be differentiated by the location of the pain, tenderness, resistance over the gall-bladder, and its radiation to the back. A tumor in the gall-bladder region may be present, sometimes jaundice, but jaundice in a large majority of cases is absent in gall-bladder inflammation. It occurs when the common duct is occluded by a stone or the result of gall-duct inflammation. Usually the pain is more intense than in appendicitis.

Perforative Peritonitis.

This condition may be due to perforation of any viscus, including the appendix. There is collapse, general abdominal tenderness, pain and tense abdominal walls; the knees are drawn up; the pulse and respiration are rapid; the temperature varies.

Gastric and Duodenal Ulcer.

These conditions usually have a symptomatology of their own—hyperacidity, pain recurring long after eating—just when the gastric juice begins to flow over the ulcerated area, relief after taking food. However, the first attack of pain may be due to a perforating ulcer. The pain is not usually so severe as in appendicitis, but may be; the resistance and tenderness are usually above the umbilicus; there may be occult blood in the stool (this cannot be ascertained at the first visit); there is no leukocytosis.

Hyperacidity.

I mention this here because I believe many of the supposed cases of simple hyperacidity are in reality gastric or duodenal ulcers, and should not be confounded with that disease or with appendicitis.

Acute Gastritis—Enteritis.

In acute gastritis and enteritis there may be fever and tenderness over the abdomen, but the tenderness and pain are diffuse; there is no leukocytosis. There is diarrhea in enteritis which may occur in appendicitis, but this is not usual.

Intestinal Obstruction.

A hernia may be present unnoticed or unknown to the patient. Look in the groins and umbilical region for a constricted mass. Usually vomiting is present and is often extremely severe and continuous. There is more collapse in volvulus, hernia and intussusception than there is in appendicitis; the tenderness may be anywhere over the abdomen. In intussusception there may often be found a mass palpable by rectal examination, especially in children. There is no leukocytosis.

Lead Colic.

Always look at the gums—*a blue lead line means lead poisoning.* More than one patient has been prepared for operation when a blue line has been found. Find the line *first.* There is obstinate constipation, the pain is continuous, vomiting is not persistent. There is no leukocytosis, but the red cells show stippling. Appendicitis existing in a patient suffering from lead poisoning will be indicated by fever, leukocytosis, local tenderness and pain plus the signs of lead poisoning.

TYPHOID FEVER.

Usually there is a history of illness continuing some time before the attack of pain, but typhoid fever symptoms often begin suddenly, especially in children. There may be tenderness, extreme in the right iliac fossa; there may be a mass simulating a mass in the appendix which may be due to enlarged pericecal glands. Sometimes there is a true appendicitis with the typhoid fever, when there is extreme difficulty in diagnosis, for all the signs of appendicitis are present, including leukocytosis. Here the presence of a Widal reaction and a history of diarrhea, rose spots, enlarged spleen will make the diagnosis. In uncomplicated typhoid there is no leukocytosis, but often leukopenia.

PNEUMONIA OR DIAPHRAGMATIC PLEURISY.

This condition must always be thought of when looking for appendicitis. If this is not done, the symptoms will deceive the very elect. There is sudden pain, tenderness and resistance in the abdomen with leukocytosis. However, in pneumonia while the pain is complained of in the abdomen there is less of the characteristic tenderness and usually less resistance; the writer has seen a case of pneumonia with resistance as great as in any peritonitis. The respiratory rate is increased. Careful physical examination will usually reveal some abnormality of the lung on the right side. The breathing is often suddenly checked by the pain; there is slight dullness over the base of the lung; the breath sounds may be fainter and not louder on the affected side. Careful examination will usually settle the question.

ACUTE PANCREATITIS.—Acute pancreatitis may be extremely difficult of differentiation. There is pain, tenderness, fever or collapse; the latter usually more marked than in appendicitis; the tenderness is farther up in the abdomen; the epigastrium is frequently extremely tender.

ACUTE EPIDIDYMITIS.

Right-sided epididymitis gives rise to such severe abdominal pain, fever and leukocytosis, that an abdominal operation may be contemplated. One glance at the testicle will make the diagnosis. I have seen a patient prepared for appendiceal operation who was suffering from epididymitis.

CARCINOMA.—Carcinoma has a long-standing history of ill health. There is frequently a tumor but no leukocytosis unless there is an inflammatory condition.

Tubercular Peritonitis.

Tubercular peritonitis has also a long-standing history. There is distention of the abdomen, or marked retraction; the tenderness is general rather than local, though occasionally the beginning may be represented by severe local pain and tenderness; there is leukocytosis. Usually tubercular lesions may be diagnosticated elsewhere in the body.

Gastric Crisis of Tabes dorsalis.

This may be indicated by severe pain in the epigastrium, with collapse and vomiting. Examination of the pupils will show Argyll-Robertson pupils. There is no patellar reflex, no leukocytosis, no fever, and no abdominal tenderness or resistance.

Dietl's Crisis.

Dietl's crisis may be mistaken for appendicitis because of the pain, tenderness, and vomiting. There is no fever, no leukocytosis. A kidney may be palpated on the right side which is frequently tender and often enlarged.

Renal Colic.

In renal colic (on the right side) there is pain and vomiting; also there is bloody urine. When the appendix dips down into the pelvis it may give these signs. In renal colic the pain is excruciating and continuous. There is no fever, and no leukocytosis. The writer has seen a case where all the signs of renal colic were present at the first visit, without fever or leukocytosis; in a very few hours the fever and leukocytosis of appendicitis appeared. Operation proved appendicitis.

Extra-uterine Pregnancy.

In this there may be acute abdominal pain and resistance low down on the right side; the pain and tenderness, however, are usually lower than in appendicitis. There is always a history of deranged menstrual function, either delay or irregular continued flow; usually there is marked anemia. Vaginal examination shows a tender mass to one side of the uterus. Fever and leukocytosis must not deceive; these are present in extra-uterine pregnancy.

Cases of **salpingitis** and appendicitis may closely resemble each other where the appendix is low or the tube high. There is fever, pain, resistance, leukocytosis, but vaginal examination should make the diagnosis. Menstrual disorders should be distinguished by the fact that there is no fever, no leukocytosis, and by the history of the case.

Toxemia of pregnancy is constantly accompanied by severe pain, especially in the epigastrium. It is mentioned here to warn against a diagnosis without thought of the possibilities of this toxemia. The urine usually contains albumin.

ACUTE INDIGESTION—BILIOUSNESS.

Acute indigestion does exist and the diagnosis may be made when all the previous conditions are excluded. Vomiting of undigested food is not sufficient cause for a diagnosis of this condition alone. When all the symptoms end by emptying the stomach there is reason to suppose the condition is due to the food ingested.

Biliousness is an antiquated term without definite signs or symptoms.

4. Intestinal Obstruction

Origin.—The symptoms of this condition depend upon whether the construction is complete or partial. The symptoms are either originally acute, or there are acute symptoms supervening upon a chronic partial obstruction. *The condition is really a symptom of various diseased conditions,* among which may be mentioned hernia, twists of the intestine, intussusception, carcinoma of the intestinal tract, obstruction by bands of adhesions—the result of disease or operation—contractures of the gut after ulceration, foreign bodies which have been swallowed or which have entered the intestine by perforating it, such as gall-stones, and which have occluded the gut.

Symptoms.—The symptoms of the complaint are abdominal pain, complete inability to have a fecal bowel movement, vomiting, abdominal distention, and in the early stages violent peristalsis, to be later followed by paresis of the gut. In the late stages the vomiting becomes fecal in character.

Conditions to be Differentiated from Intestinal Obstruction

This may be mistaken for:

Peritonitis
Acute dilatation of the stomach
Atony of the intestine
Constipation.

PERITONITIS.

From peritonitis the diagnosis is often difficult and sometimes impossible. In peritonitis usually there is entire absence of peristalsis; never is there the violent peristalsis found in the early stages of obstruction. Pain is more general, more diffuse, and the abdomen is much more tender in peritonitis than in obstruction.

Vomiting of obstruction soon becomes fecal, while in peritonitis there is the sudden expulsion of large amounts of greenish material, evidently composed of disorganized stomach contents; sometimes mouthfuls of this same greenish material are spat up. The distention of obstruction while it may be *general* is sometimes *localized*, and when such is the case, localized distention argues against general peritonitis.

Search for any of the causes of obstruction is imperative if failure is not to be the rule in diagnosis. The finding of a rational reason for obstruction is good evidence of its presence and against peritonitis.

Leukocytosis is the rule in peritonitis. It may be present in obstruction if local peritonitis has supervened or if gangrene of the intestine has occurred.

Acute Dilatation of the Stomach.

This often follows operations and is the result of overeating, or it may occur during the course of certain infections—particularly pneumonia. There is obstinate constipation, the stomach is greatly distended as evidenced by the tympanitic prominence in the epigastrium. There is usually a succussion splash over the stomach. The stomach tube will at once remove the distention, if it is due to stomach dilatation but will have little effect in the distention of the intestines from peritonitis. The patient is frequently cured of dilatation of the stomach by the use of the tube. There is little effect in peritonitis.

Atony of the Intestines.

Atony of the intestines is chronic in character, there is little pain, no fever and great emaciation.

Constipation.

Constipation may resemble certain cases of obstruction. There are cases of constipation in elderly persons where acute symptoms supervene upon a chronic constipation; this latter may be due to a gradual closing of an old obstruction. Only extreme care to discover an organic cause will avoid disaster. If repeated high enemata fail to bring about a fecal movement the inability to have a bowel movement is not the result of a simple constipation, but is organic in origin. An x-ray examination is here of value.

5. Constipation

This condition is always a *symptom.* Its diagnosis is extremely easy. Difficult bowel movements separated by one or more days, with hard stools, are the points upon which the diagnosis depends. To separate the symptoms which are due to simple inability of the intestine to empty itself from those due to an organic cause is often difficult.

One must here consider stricture of the intestine from any cause, atony of the intestine, malposition, dilatation of the colon, and some acute condition from the functional state.

Signs.—In certain chronic conditions, such as gradually narrowing stricture due to adhesions, or a slow narrowing due to scirrhous cancer, especially of the lower bowel, the symptoms are those of a gradually increasing constipation. Therefore, any case of chronic constipation should be carefully examined for signs of obstruction before any ordinary treatment for constipation is given. As a matter of fact, increasing constipation following an operation, a history of an abdominal inflammatory condition, or in an individual of advanced age, even without other symptoms, is to be looked upon with the greatest suspicion. Local tenderness and pain, increased peristalsis over the seat of constriction, are the signs most to be relied upon to distinguish the first two conditions, while a rectal examination or examination with a proctoscope is the most useful help. In carcinomatous cases a radiograph will show here, as well as in dilatation of the colon, the exact condition existing.

Conditions to be Differentiated from Constipation

Acute constipation is likely to be mistaken for a simple acute constipation, acute appendicitis, acute obstruction caused by incarcerated hernia, volvulus and intussusception. All of these conditions have their especial symptoms.

ATONY.

Atony may be distinguished by the general condition of systemic failure, by the monstrous abdominal distention and the thin belly walls.

HERNIA.

This can be demonstrated by a palpable mass at the inguinal ring, femoral ring, or at the umbilicus. There is always pain accompanying the symptoms.

VOLVULUS AND INTUSSUSCEPTION.

These may occur suddenly. There is pain, distention of the abdomen and collapse—all are wanting in constipation.

APPENDICITIS.

In appendicitis there is fever, local tenderness and leukocytosis.

The symptoms of constipation will be relieved by a bowel movement.

None of the other conditions mentioned will disappear, and often the signs are made much worse by purgation; hence purgatives must never be given unless organic conditions can be excluded.

6. Visceroptosis

(*Splanchnoptosis, Enteroptosis, Glenard's Disease*)

This is a condition in which the position of all the abdominal organs is lower than normal. We will consider *the symptoms referable to the general condition* first, and later the *symptoms referable to the single organs and their differentiation from conditions with somewhat similar symptoms.*

Frequently most of the abdominal organs are ptosed without symptoms of any kind; sometimes, however, the patient may be neurasthenic. Neurasthenia, therefore, calls for a careful abdominal examination. Often there will be found a ptosis of one or more of the abdominal organs; this may act as an exciting cause of the neurasthenia. Care must be exercised, however, in ascribing the neurasthenic symptoms to the ptosis without taking the history of the case and a thorough, general physical examination into consideration.

Differentiation.—Gastric and intestinal ptosis may simulate actual organic disease of the stomach and intestines, even carcinoma being caricatured by this condition. Differentiation can be made by careful physical examination and attention to laboratory methods—above all by use of the x-ray. An x-ray picture taken with a bismuth meal will positively show the positions of the stomach and intestines.

In CARCINOMA OF THE STOMACH there may be a history of long-standing indigestion caused by conditions other than the carcinoma; with the appearance of carcinoma proper there is loss of weight; and gastric contents are characteristic of that condition; there may be blood in the stools. GASTRIC ULCER differs in that the pain and symptoms of this condition have a curious and characteristic periodicity and chronicity, while this is only simulated by ptosis and lacks the systematic regularity of the pain of ulcer.

ENTERITIS may be simulated by the vague abdominal pains of ptosis and sometimes by diarrhea. Indeed the malposition of the viscera may cause an actual enteritis.

CHOLELITHIASIS, CHOLECYSTITIS and CHOLANGITIS may all be simulated by ptosis of the stomach and intestines—by local pain, even by distress and distention of the gall-bladder and jaundice, the latter caused by kinking of the cystic and common ducts.

Great care must be taken, to take into consideration the *position of the stomach* in all cases simulating the above condition, when operation is contemplated. On the other hand, a case of GASTROPTOSIS may be complicated by a gastric ulcer or by gall-stones and the symptoms of the latter conditions may be wrongly attributed to the gastroptosis existing. Any case of gastroptosis with symptoms of ulcer or gall-stones which persist in spite of treatment should be subjected to a thorough and painstaking x-ray for ulcer and gall-stones to be followed by an exploratory laparotomy if the plates are suspicious.

RENAL CALCULUS, HYDRONEPHROSIS AND OTHER TUMORS OF THE KIDNEY may be simulated by ptosis of the kidney, whenever the movement of the kidney is extensive enough to allow of kinking of the ureter; renal pain and renal colic may occur. Here urine examination, the x-ray, and if necessary cystoscopic examination, with examination of the ureters and position of the kidneys, will make the diagnosis certain.

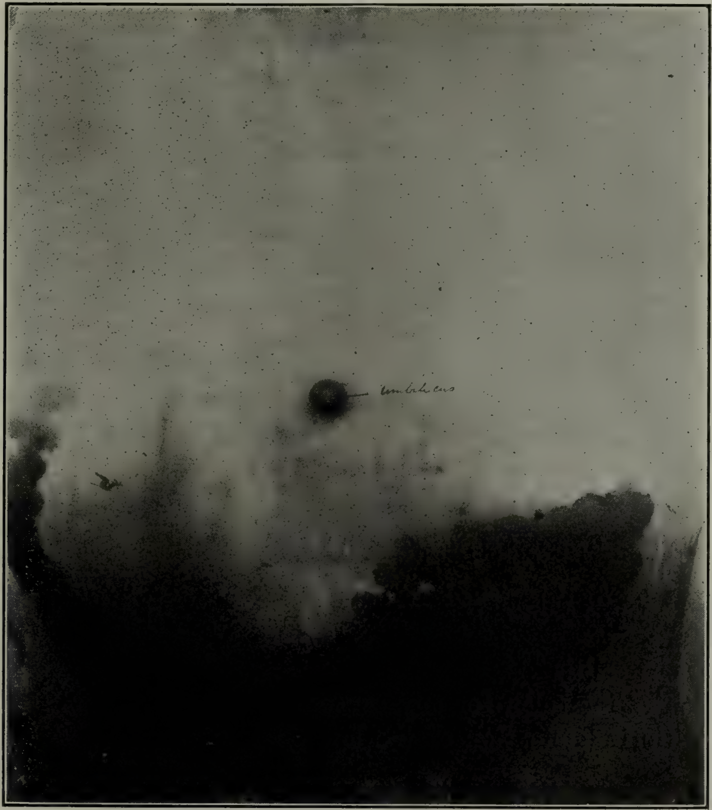


Fig. 56.—Extreme Visceroptosis—Shadow is Large Intestine in the Pelvis.

Ptosis of the cecum alone, CECUM MOBILE, will frequently simulate APPENDICITIS by pain and local tenderness. Blood examination, however, will help in the diagnosis. Fever and leukocytosis which are characteristic of appendicitis are wanting in cecum mobile. While there is local tenderness and sometimes resistance in cecum mobile, it lacks the peculiar spasm of the abdominal wall present in appendicitis.

Mobility of the liver may be mistaken for tumor or other enlargement of that organ, but the extreme mobility of the liver with its normal size, as ascertained both by percussion and palpation, will serve to make the distinction.

A movable spleen may be mistaken for a new growth or for a movable left kidney; the author reported such a case. Usually the shape of the spleen can be so well outlined that an error is unlikely.

Dilatation of the stomach may be simulated by ptosis of that organ. Gastric lavage and an x-ray picture will show the marked difference of the two conditions (Fig. 56).

7. Mucous Colitis

This is a chronic condition characterized by neurasthenia, with constipation or more or less frequent bowel movements, consisting sometimes of normal stools, sometimes of normal stools covered with mucus, and sometimes of mucus alone. Cramplike pains are common either just before a stool or between stools.

The mucus is thick and glairy in character and sometimes forms cylindrical masses as though it were a cast of the entire coat of the bowel. Frequently the masses are rolled into wormlike bodies and may, by the laity, be mistaken for parasites.

Conditions to be Differentiated from Mucous Colitis

This must be distinguished from:

Parasites
Membranous colitis
Ulceration of the colon for any cause
Diphtheritic inflammation of the lower bowel.

Parasites.

Parasites are easily distinguished by the aid of the microscope, the presence of ova, or the worm itself. The microscope shows the "worms" which annoy the laity to such an extreme to be bits of harmless mucus. There is no excuse for a delay in this differentiation.

Membranous Colitis.

Membranous colitis can also be distinguished by means of the microscope by the fact that here there are many leukocytes in the meshes interlacing strands of fibrin. If the membrane is due to Klebs-Löffler bacilli, the condition is of necessity much more acute in its course, and the bacilli can be demonstrated in the membrane. The membrane may be due to infection by pneumococci.

ULCERATION OF THE BOWEL.

This is characterized by the discharge of blood with pus and mucus, and if the ulcer is situated in the lower bowel, it can be demonstrated by the use of the proctoscope.

8. Simple Colitis

Etiology.—This form of inflammation of the colon is distinguished from that due to specific diseases, such as dysentery, or that which is secondary to some systemic disease, such as chronic nephritis. It occurs suddenly with fever, distress, burning about the rectum, tenesmus, frequent stools, often containing blood and mucus. It is the result of some indiscretion of diet and seems to follow overexertion and exposure in certain instances.

Since the observations of Duval, Flexner and others, many cases which were formerly considered *acute simple colitis* are properly classed as *true dysentery* due to infection by one or the other strain of dysentery bacilli. The symptoms of the two conditions are almost exactly the same.

Conditions to be Differentiated from Simple Colitis

Dysentery
Malignant disease of the colon or rectum
Hemorrhoids
Foreign bodies impacted or lodged in the intestine
Intussusception.

DYSENTERY.

This condition can be distinguished by its general symptoms, fever, great prostration, by the greater tendency to toxic complications such as nephritis, and above all by the ability to demonstrate dysentery bacilli or amebae in the injecta.

MALIGNANT DISEASES OF THE COLON AND RECTUM.

This is a more chronic condition. It is characterized by the following symptoms: great loss of weight and blood; sudden signs of obstruction are apt to occur; a palpable mass is likely to be present, to be demonstrated through the abdominal wall or through the rectum.

Hemorrhoids.

Hemorrhoids may be characterized by blood and mucus in the stools. Ocular inspection will make the diagnosis.

Intussusception.

This condition at first may have bloody mucus and stools, but the frequent stools soon are replaced by complete obstruction of the bowels—this does not occur in colitis.

9. Intestinal Sand

Intestinal sand is a true mineral inorganic substance consisting of calcium, phosphorus, carbon, magnesium and iron, which is discharged from the bowel. It has all the characteristics of ordinary sand. Sometimes it is red, sometimes whitish.

It may be mistaken for urates when the stool has been contaminated by urine. Care must be taken that an hysterical patient does not attempt to supplant the ordinary sand of the street which has been mixed with the stool for the purpose of deception. It may also be confused with a gritty substance due to vegetable residue. Here the microscope will show the material to be of vegetable and not mineral matter.

10. Dilatation of the Colon

Dilatation of the colon is characterized by great abdominal distention, by pain, and by obstinate constipation.

The condition is either *congenital* or *acquired.* The latter form is the result of atony of the wall of the bowel, or the result of a long-standing, slowly contracting stricture of the bowel.

Conditions to be Differentiated from Dilatation of the Colon

The congenital form is frequently mistaken for INTESTINAL ATONY, and the acquired for SIMPLE CONSTIPATION; it is also mistaken for dilatation of the stomach. The condition can be successfully diagnosed by a bismuth meal and an x-ray. It is only by this relatively recent method that the dilatation can be certainly differentiated from the various diseased conditions with which it may be confounded.

Dilatation of the Stomach may be diagnosed by careful removal of the stomach contents and by then refilling the stomach with air through the stomach tube. If the distention persists it is probably intestinal in character. Again, the colon may be filled with air from the rectum, which, if the distention is in the colon, will make the tension more extreme.

11. Diverticulitis

This name should apply to inflammation of a diverticulum of the bowel in any portion of the intestinal canal. However, the special term has become attached to inflammation of the diverticuli which are frequently present in the descending colon, and especially in the sigmoid flexure. Here the diverticulum which is frequently small becomes occluded, the wall inflames, becomes adherent to the surrounding tissues and forms a decided mass. All the signs of suppuration may occur—local tenderness, fluctuation, leukocytosis, and fever.

Diagnosis.—It is often mistaken for carcinoma of that portion of the gut, particularly when it makes its appearance in an individual past middle age. The increasing size of the mass, the pain, the possible emaciation and constipation help to strengthen this opinion. As a rule, however, carcinoma has a more chronic course and there is more emaciation.

Removal of the mass by excision and its examination is not only the sole means of making a diagnosis, but is the proper method of treatment.

A fecal mass might be mistaken for a tumor due to diverticulitis, but here the mass resembles that found in impactions anywhere. It can be indented, and after careful use of enemeta and laxatives it disappears.

Cancer or other new growths in the gut are slow in their onset. Usually cachectic symptoms occur, and obstruction of the bowel is more common than in diverticulitis.

Diverticulitis may give rise to a purulent peritonitis, the result of infection of the diverticulum. The only means by which such a peritonitis can be traced to its source is the discovery and diagnosis of the diverticulum and the recognition of it as the starting point of the peritonitis.

A displaced kidney or floating spleen might simulate a diverticulitis, but there is much less likelihood of either taking on a suppurative condition. The author, however, reported in Univ. M. Mag., 1890, a case in which a wandering spleen became incarcerated in the pelvis and gave rise to signs of local inflammation.

12. Affections of the Mesentery

Cysts, diseases of the vessels and hematoma occur.

They cannot be diagnosed during life—with the possible exception of embolism of the mesenteric arteries.

In the presence of heart disease, or sclerosed vessels, or a septic infection, sudden abdominal pain with rapidly appearing symptoms of intestinal obstruction, might be considered as due to an embolus.

H. Diseases of the Liver

1. Jaundice

Jaundice is a yellow discoloration of the skin, mucous membrane and secretions, due to obstruction in some portion of the bile ducts, either in the capillaries or in the larger ducts themselves. Even jaundice due to blood destruction is obstructive in character, the thickened and disturbed blood in the capillaries of the liver causing obstruction of the terminal bile ducts (Plate 5, Fig. 1).

Besides the yellow discoloration of the skin, sclera and mucous membranes in general, there is general itching over the whole skin.

There are clay-colored stools and high-colored urine.

A history of gall-stones enlarged and tenderness of gall-bladder tend to establish the diagnosis. In severe jaundice bradycardia may be present.

Conditions to be Differentiated from Jaundice

It must be differentiated from:

Addison's disease
Splenic anemia
Vitiligo
Pigmentation in anemia
Pigmentation due to scratching
Vagabond disease
Sallow cachectic discoloration.

ADDISON'S DISEASE.

Addison's disease is often characterized by a discoloration most marked in exposed portions of the skin, varying from light yellow to dark brown, and accompanying extreme athenia and rapid feeble pulse (Plate 5, Fig. 3).

SPLENIC ANEMIA.

Pigmentation as part of the symptomatology of splenomegalia is common and might be mistaken for jaundice. Here the large spleen, the absence of discolored mucous membranes and urine and the absence of clay-colored stools will help in the diagnosis.

VITILIGO.

Vitiligo, characterized as it is by dark patches interspersed with loss of pigment, might possibly be taken for jaundice, but it may be distinguished from jaundice by the fact that in the latter the color is uniform, while in vitiligo patches are found which are entirely free from pigment of any character. In vitiligo there is no sign of general disease; it is also much more chronic than any form of jaundice.

The term "liver spots," given by the laity to vitiligo, is entirely erroneous. The discoloration has no relation to the liver.

THE PIGMENTATION OF ANEMIA.

This condition may be distinguished by examination of the blood, for while a jaundiced individual may be extremely anemic, there is wanting the more or less characteristic blood picture due to one of the serious anemias; also, the symptoms of the characteristic anemias are wanting in simple jaundice. Again, it must be remembered that the more or less yellow tint of the skin in pernicious anemia is not a jaundice; there is no discoloration of the urine and there are no clay-colored stools (Plate 5, Fig. 2).

PIGMENTATION DUE TO SCRATCHING—VAGABOND DISEASE.

Pigmentation due to scratching, from filthy conditions of skin and from vermin, may be quite extreme, but it never affects the sclera; the stools and the urine are normal; there is the history of vagabondism or careless habits.

Hereditary Hemolytic Jaundice

This condition occurs soon after birth, appearing in more than one child of the same family. There is often syphilis in the father or mother. There is often stenosis of the duct with angiocholitis.

The writer has knowledge of three children afflicted by this condition, having seen the third child; all of these children developed jaundice on the third or fourth day after birth. In these cases there were subcutaneous hemorrhages, in one valve hemorrhage from the mucous membranes, in another there were subdural hemorrhages which caused hemiplegia. The last case recovered through the injection of human blood serum.

2. Echinococcus Disease of the Liver

Origin.—This condition is due to the lodgment of the larva of Tenia echinococcus in the liver and its development there.

Symptoms.—Its symptoms are thus described in Tyson's "Practice of Medicine," 1913, edited by Fussell:

> "Small cysts may occasion no symptoms, being often unexpectedly found at necropsy, and under any circumstances the failure of health is very gradual at first. As cysts become large they produce a sense of weight or dragging in the region of the liver, and other symptoms depending upon their size and situation; jaundice if they cause obstruction of the biliary passages; dyspnea and cardiac

disturbance if they encroach on the heart or lungs; pyemic symptoms—that is, fever, sweat, and sometimes chills, with rapid exhaustion if they suppurate. The liver may become very much enlarged, demonstrable by inspection, palpation, and percussion. If there is a single superficial cyst either in the right or left lobe, it may be felt either as an elastic or fluctuating tumor; or there may be the distinct feel of nodular growth over the liver. If posterior in the right lobe, it may encroach on the inferior part of the lung and pleural space, causing dullness on percussion posteriorly and posterolaterally, and other signs of pleuritic effusion. Hydatid thrill or fremitus is always to be sought for. It may be found, if the cyst is superficial, by placing one hand over the tumor and tapping lightly with the fingers of the other. The result is a vibrating or trembling movement felt for a short time. It is not often obtainable, and is possible only with superficial cysts. It has been described by Briancon to the collision of the daughter cysts.

If rupture occurs other symptoms are added. The pleural cavity is often invaded, or the lungs, as evidenced by the expectoration of cysts and hooklets; the bile passages by the presence of jaundice or increased jaundice; and the subsequent appearance of hooklets and cysts in the fecal discharges. Rupture into the stomach is manifested by the vomiting of hooklets and cysts; into the vena cava, by embarrassment of the cardiac action and pulmonary thrombosis from lodgment of cysts; into the pericardium, by fatal pericarditis; into the peritoneum, by fatal peritonitis; and into the abdominal wall, by outward discharge."

Conditions to be Differentiated from Echinococcus of the Liver

Echinococcus of the liver can be mistaken for:

Cancer
Carcinoma
Syphilis
Cirrhosis of the liver
Hydronephrosis
Dilated gall-bladder.

CANCER.

Cancer is of much shorter duration; there is much more loss of weight; the liver is uniformly enlarged with umbilicated nodules. Carcinoma in other portions of the body, especially in the uterus and rectum, will lead to the diagnosis.

SYPHILIS.

In syphilis there is a Wassermann reaction, tendency to ascites, and improvement in many cases by the use of salvarsan and other antisyphilitic remedies.

CIRRHOSIS OF THE LIVER.

Cirrhosis has the history of alcoholism, persistent ascites and enlarged spleen.

HYDRONEPHROSIS.

Hydronephrosis may be simulated when the cyst is in the right side encroaching on the renal region. There is lack of urinary disturbances in echinococcus of the liver. Catheterization of the ureters will show the catheter toward the pelvis of the diseased kidney; it could not be seen connected with a hydatid cyst of the liver.

DILATED GALL-BLADDER.

A dilated gall-bladder is usually mobile. Inspection by incision (the proper method) would not show hooklets and would demonstrate the condition of the gall-bladder.

3. Icterus neonatorum

Occurrence.—Icterus neonatorum occurs in newborn infants in many cases simply as a discoloration of the skin, giving no other symptoms. According to Holt, it occurs in about one-third of the cases.

Cause.—Severe and fatal icterus occurs in infants as the result of syphilis, and of occlusion or absence of bile ducts.

Conditions to be Differentiated from Icterus neonatorum

This condition must be separated from:

Hemorrhages in the newborn
Jaundice occurring as the result of septic infection of the umbilical cord
Hereditary icterus.

HEMORRHAGES IN THE NEWBORN.

Hemorrhagic disease of the newborn is recognized by the presence of hemorrhage from the stomach and bowel, which is often rapidly fatal.

JAUNDICE AS RESULT OF SEPTIC INFECTION OF UMBILICAL CORD.

Sepsis of the umbilical cord is distinguished by fever, rapid anemia and leukocytosis. The external cord is often seen to be diseased.

HEREDITARY ICTERUS.

Hereditary jaundice occurs in *various children of the same parents.* It is accompanied by fever, often by petechia, and by hemorrhages from the mucous membranes.

4. Acute Yellow Atrophy of the Liver

(*Malignant Jaundice—Icterus gravis*)

Occurrence.—Lately it has been learned that necrosis of the liver cells, decrease in size of the liver, jaundice and toxemia are present in other conditions, and are apparently exactly the same as are present in phosphorus poisoning. These conditions are found in certain infections, such as syphilis, diphtheria and typhoid fever. They may occur in pregnancy and after chloroform administrations.

Symptoms.—The symptoms are rapid jaundice, possibly preceded by gastro-intestinal symptoms and followed by severe toxic symptoms—headache, vomiting, rapid pulse, hemorrhages under the skin and coma. The patient rapidly becomes extremely ill, with rapid pulse, dyspnea and lethargy, the symptoms soon becoming typhoid in character. Delirium is common; the urine is bile-colored and may contain leucin and tyrosin crystals; there is always albumin.

Conditions to be Differentiated from Acute Yellow Atrophy of the Liver

It may be mistaken for:

Catarrhal jaundice
Hypertrophic cirrhosis
Phosphorus poisoning.

CATARRHAL JAUNDICE.

Catarrhal jaundice is short in its course and recovers; no given case of any severity, however, can be safely differentiated in the beginning from a severe form. Catarrhal jaundice may be accompanied by very severe symptoms, although it is usually a mild condition. The liver is usually enlarged; it often follows a drinking bout. Leucin and tyrosin do not appear in the urine. In very severe attacks of jaundice and fever with toxic symptoms which complicate portal cirrhosis, one may doubt the character of the conditions, but the absence of tyrosin and leucin and the history of cirrhosis will help to decide in favor of infection during cirrhosis.

HYPERTROPHIC CIRRHOSIS.

Hypertrophic cirrhosis is chronic in its course; the liver and spleen are enlarged. Leucin and tyrosin do not occur.

PHOSPHORUS POISONING.

Phosphorus poisoning is said not to be accompanied by leucin and tyrosin in the urine. Only a distinct history of poisoning by phosphorus will make the diagnosis even at post mortem.

5. Hypostatic Congestion of the Liver

Origin.—This condition is a part of the symptom complex and physical signs of cardiac decompensation, whatever the cause of the decompensation may be. Hence it is found in all cases of severe cardiac valvular disease where there is decompensation, and in emphysema, arterial hypertension, etc., where the back pressure upon the heart is so great that the venous circulation is overfilled, the liver becoming enlarged because of engorgement with blood.

Symptoms.—The signs are enlargement and tenderness of the liver, this organ frequently being felt as far down as the umbilicus. All other symptoms and signs of cardiac failure, to wit, dyspnea, cough, cyanosis, palpitation, enlargement of the heart and edema of the extremities, may accompany the condition.

Passive Congestion of the Liver.—With these symptoms we are probably dealing with a simple passive congestion of the liver. Any enlargement unaccompanied by cardiac failure is either an actual cirrhosis due to previous and long-standing heart disease or it is due to some disease of the liver unconnected with the circulation; in a word, *to make a diagnosis of passive congestion of the liver, cardiac failure is necessary.* To be certain one is not dealing with a primary condition of the liver with a secondary cardiac failure one must be sure that the history does not give an enlargement of the liver before signs of cardiac failure are present.

In practically all cases of passive congestion there develops sooner or later a cirrhosis which becomes an integral part of the disease. In cases where the tricuspid regurgitation is very marked, there is a true expansile pulsation of the entire liver. This must be distinguished from a transmitted pulsation which is due either to the underlying aorta or to impact with the enlarged right heart.

Active Congestion of the Liver.—Active congestion of the liver is the result of overeating, of various acute infections and of certain poisons, and is common in Europeans in the tropics. All of these conditions are acute; there is entirely wanting the chronic cardiac decompensation necessary to a diagnosis of passive congestion.

Conditions to be Differentiated from Hypostatic Congestion of the Liver

Portal cirrhosis
Biliary cirrhosis
Carcinoma of liver
Amyloid liver
Syphilis of liver
Leukemia.

PORTAL CIRRHOSIS.

A case of portal cirrhosis, with enlargement, is indicated by the fact that the patient is an alcoholic; there is engorgement of the entire portal system, frequently accompanied by vomiting of blood; there may be attacks of fever accompanied by increasing jaundice. There is no cardiac failure except toward the end of the course of the cirrhosis. The spleen is enlarged.

BILIARY CIRRHOSIS.

This is a disease extremely chronic in character. There is fever, continuous jaundice, with enlarged spleen and liver.

CARCINOMA OF THE LIVER.

Carcinoma of the liver can be differentiated by the loss of weight and of strength, by new growths in the liver itself, and by the absence of cardiac decompensation.

AMYLOID LIVER.

In amyloid liver there are no signs of primary cardiac failure; syphilis, tuberculosis or long-standing suppuration are causative factors.

SYPHILIS OF THE LIVER.

In syphilis of the liver, the organ is apt to be irregular in shape; there is a history of syphilis. The Wassermann reaction may be present. The therapeutic test is of great value.

LEUKEMIA.

In leukemia the increase of leukocytes and enlargement of the spleen and lymphatic glands distinguish it from hypostatic congestion.

6. Acute Catarrhal Cholangitis

(Acute Catarrhal Jaundice)

This condition is characterized by the sudden appearance of jaundice, usually first noticed as a discoloration of the sclera, gradually increasing in intensity and extent until the whole body is a lemon yellow color. The urine becomes bile-stained. It is frequently accompanied by digestive disturbances, vomiting, diarrhea and epigastric pain.

Acute catarrhal jaundice may occur at any age, but when jaundice persists, and especially in an individual over thirty years of age, either with or without symptoms, one must wait for a certain differentiation before a diagnosis of catarrhal jaundice is made. Jaundice, in which the symptoms last more than two or three weeks, may have some other cause than the simple catarrh of the bile ducts.

Conditions to be Differentiated from Catarrhal Jaundice

Obstructive jaundice.
  The obstruction may be due to—
  (*a*) Gall-stones
  (*b*) Kinking of the common or hepatic duct
  (*c*) Pressure from enlarged glands upon the common duct
  (*d*) Pressure from other tumors
  (*e*) Cancer of the bile ducts
  (*f*) Suppurating process of the bile ducts
The action of poisons of various kinds
Abscess of the liver
Cancer of the liver.

GALL-STONES.

Gall-stones may be differentiated by the age of the patient. The occurrence of gall-stones is rather rare before the age of thirty-five, though they may occur at any age. Pain is more frequent and severe than it is in catarrhal jaundice.

Prolonged jaundice is indicative of gall-stones rather than of catarrh of the bile ducts. Intermittent fever, chills and sweats, followed by jaundice, indicate a movable (ball valve) stone in the common duct. Prolonged tenderness is much more common in gall-stones. When the jaundice is preceded by severe pain, and pain and tenderness continue in the region of the gall-bladder, the cause is more likely to be due to a stone.

KINKING OF THE COMMON HEPATIC DUCT.

Ptosis of the stomach or the liver, when accompanied by jaundice and without symptoms of gall-stones, indicates a kinking of the common duct. The jaundice is more prolonged than when catarrhal in character, and is relieved by a properly applied bandage.

PRESSURE FROM ENLARGED GLANDS UPON THE COMMON DUCT.

Enlarged glands about the common or hepatic duct constantly give rise to a persistent jaundice. In the absence of the signs of gall-stones and the absence of a palpable tumor one may suspect the presence of enlarged glands.

Pressure from Other Tumors.

A palpable tumor of the upper abdomen—whatever the character of this tumor may be—when it is accompanied by jaundice, may be taken as causing the jaundice by pressure.

Cancer of the Bile Ducts.

Cancer of the gall-bladder and gall-ducts is apt to be accompanied by enlargement of the gall-bladder. There is persistent pain, loss of weight and strength.

Suppurating Process of the Bile Ducts.

Suppurative and infective cholangitis is recognized by severe constitutional disturbances, leukocytosis, fever sweats, chills, and by persistent and severe pain over the gall-bladder region. The presence of this form of cholangitis may be suspected when there has been previous inflammation in the region of the appendix.

Jaundice Due to Poisons.

Pneumonia and typhoid fever occasionally are accompanied by jaundice. Frequently this jaundice is due to a hemolysis, resulting from the toxins circulating in the blood. Injected poisons, such as phosphorus, are apt to cause jaundice. These conditions must be diagnosed by the presence of their systemic symptoms.

Abscess of the Liver.

This is secondary to suppuration of the bile ducts, a pylephlebitis or to a preëxisting amebic dysentery. The suppurating of the bile ducts is suggested by the severe character of the disease which has preceded the jaundice either in the form of an appendicitis or cholangitis, and differs greatly from the mild symptoms of catarrhal jaundice. Solitary abscess of the liver is indicated by enlargement of the liver or the history of a previous dysentery and by the severe illness of the patient.

Cancer of the Liver.

Cancer of the liver is recognized by gradual enlargement of the liver, with a previous history of digestive disturbance continued over a long time, and by the appearance of nodules over the liver—a picture which differs in all respects from that of catarrhal jaundice.

7. Cholangitis Not Due to Gall-stones

This condition is either severe or mild in its symptoms, probably depending upon the activity of the infecting organism. It may be simply inflammation of the bile ducts due to some organism of low vitality or it may be due to a most virulent organism.

In the first instance there is slight epigastric distress, fever of mild character or intermittent in type, usually accompanied by jaundice of mild or severe degree.

In the more severe infection there is severe fever, chills, leukocytosis and jaundice, depending for its degree upon the amount of obstruction.

8. Angiocholitis

Angiocholitis is one of the results of gall-stones; when it is acute and suppurative it causes rapidly fatal condition due to multiple abscess of the liver. The suppurative form may accompany or follow acute infections; when chronic, there is occlusion of the common duct and monstrous dilatation, not only of the common duct but of the various hepatic ducts—the whole liver is channelled with dilated ducts.

The suppurative condition can be suspected when serious septic fever follows or accompanies gall-stones. There is always jaundice.

Diagnosis.—It can be differentiated from other suppurative conditions of the liver by the previous history of gall-stones and the constant presence of jaundice. It can be distinguished from jaundice accompanying other conditions, such as pneumonia and other infections, by the presence of tenderness of the liver and increase in its size, and the absence of physical signs of the other infections.

9. Acute Cholecystitis

Physical Signs.—This condition varies in its symptoms and course, depending upon the virulence of the infecting organism. They vary from a simple pain and tenderness in the epigastrium, with but few constitutional symptoms, to violent pain, great tenderness, general systemic depression rapidly terminating in gangrene of the gall-bladder and death. There is always leukocytosis.

Origin.—The severe forms are due to an infection by the various bacteria—the colon bacilli, the typhoid bacilli, pneumonococcus, the staphylococcus or streptococcus. There may or may not be the presence of gall-stones as the starting point.

The forms which are usually mild also occur in the course of infective fevers, particularly in typhoid fever, and are believed to be the direct cause of many of the relapses in this condition.

Conditions to be Differentiated from Acute Cholecystitis

The condition is most frequently mistaken for appendicitis and for perforating gastric ulcer. Indeed the diagnosis between suppurative appendicitis and suppurative cholecystitis is most difficult, and the wise physician will not wait to make any very finely drawn diagnosis but will operate upon his case at once, and will relieve the condition at whichever point it may be.

APPENDICITIS.

The points which are most likely to be of value in differentiation are firstly that in cholecystitis the pain is usually in the epigastrium, radiating to the right side over the liver, whereas in appendicitis the pain, resistance and tenderness are over the appendiceal region. The greatest difference perhaps is that, as a rule, acute cholecystitis is very much more violent in its symptoms than is acute suppurative appendicitis.

PERFORATING GASTRIC ULCER.

In perforating gastric ulcer there is usually the history of long-standing pain relieved by eating with the typical history of recurrent attacks which are not, as a rule, present in gall-bladder disease.

As mentioned in the beginning, however, the chief point is the realization of the similarity of these three conditions, often the impossibility of making a positive diagnosis, and the great necessity of an exploratory incision in order to settle the question and save the life of the patient.

10. Cancer of the Bile Passages

According to Courvoisier's law, deepening jaundice with distention of the gall-bladder, in a large per cent of the cases, is due to cancer of some part of the biliary passages.

Conditions to be Differentiated from Cancer of the Bile Passages

It might be mistaken for the following conditions, the special features of which are here given:

Impacted gall-stones
Cancer of the head of the pancreas
Tumor
Occlusion of the bile ducts.

IMPACTED GALL-STONES.

The disease is distinguished from impacted gall-stones by the fact that in gall-stones there is usually a previous history of gall-stone colic; there is, as stated, less likelihood of dilatation of the gall-bladder when the obstruction is due to stone than when it is due to cancer; there is less loss of weight when the jaundice is due to stones, than in cancer. It must not be forgotten, however, that stones are often the precursors of cancer and any case which has a long-standing history of gall-stones may develop a history and symptoms of cancer.

CANCER OF THE HEAD OF THE PANCREAS.

This may cause complete obstruction of the common duct. Unless there be a palpable tumor, it is difficult to make a diagnosis.

TUMOR.

From a tumor of any kind pressing on the passages, a diagnosis can only be made by the appearance of a tumor in the abdomen.

11. Cholelithiasis

As long as gall-stones in the gall-bladder are quiescent and the wall is not infected, they are without symptoms. They are sometimes, and for the same reason, without symptoms when they are in the common duct unless their size is great enough to occlude the duct.

Acute symptoms arise from gall-stones in the form of biliary colic and inflammatory conditions accompanying the infection.

The *chronic results* of gall-stones are varied—chronic indigestion, jaundice, suppuration of the bile passage, ulceration of the bile passages, dilatation of the ducts or gall-bladder, intestinal obstruction and severe anemia being among the number.

Acute Symptoms.—Biliary colic usually occurs suddenly. There is extreme pain radiating around the body and referred to the back, under the right shoulder blade. There is no jaundice if the stone or inflammation is confined to the gall-bladder or cystic duct. Jaundice occurs only if the stone or the resulting inflammation occludes the hepatic or common duct. Many cases of gall-stones are without the symptom of jaundice. Vomiting is common—this sometimes accompanies the pain and at other times is quite continuous. The pain may cease suddenly by reason of the gall-stones receding into the gall-bladder or back into the duct or passing on into the intestine. A gall-stone may be found in the feces, but failing to find a gall-stone is not evidence that the attack was not due to cholelithiasis. Occasionally the attack of colic is so severe that death seems imminent. This may be due to simple collapse or to the occurrence of a true rupture of the gall-bladder (this accident occurred in one case in which the writer made an autopsy), or it may result from the rupture of the duct into the intestine or gall-bladder, the stone passing into the intestine. Charcot's fever or intermittent attacks of chill, fever and sweats followed by jaundice may closely resemble malarial fever, but they are followed by jaundice. They are due to absorption of septic or toxic material from the seat of the inflammation.

An x-ray made by an expert will sometimes show the shadow of a gall-stone.

Conditions to be Differentiated from Cholelithiasis

Biliary colic may be mistaken for:

Appendiceal colic
Renal colic
Pain of gastric or duodenal ulcer
Lead colic
Inflammatory conditions of bile ducts and gall-bladder
Pain due to spinal caries or other inflammatory conditions in the spinal column
Gastric crises
Pancreatitis
Angina pectoris
Pyloric obstruction
Pericarditis or pneumonia
Acute indigestion
Chronic pancreatitis
Cancer of stomach and bile ducts

Appendiceal Colic.

Appendicitis is distinguished by the presence of tenderness and resistance in the region of the appendix and the frequency of leukocytosis; it is not as a rule accompanied by jaundice.

Renal Colic.

In renal colic there is blood or pus in the urine, the pain radiates to the groin and there is usually tenderness over the kidney. An x-ray will show the shadow of a stone in the region of the kidney or ureter.

Pain of Gastric or Duodenal Ulcer.

The pain of gastric or duodenal ulcer has relation to the time of meals —mostly after meals—and is relieved by eating. There is usually hyperacidity.

LEAD COLIC.

Lead colic shows a blue line upon the gums and a degeneration of the red blood cells. In abdominal pain the gums should always be examined for a line, because the history is frequently incomplete.

INFLAMMATORY CONDITIONS OF THE BILE DUCTS AND GALL-BLADDER.

If these conditions arise there is tenderness, pain and resistance over the gall-bladder, fever and leukocytosis.

PAIN DUE TO SPINAL CARIES.

Pain of spinal caries may be differentiated by careful examination of the spinal column. Tenderness over one or more vertebrae, limitation of the movements of the spine and transference of the pain along the line of the intercostal nerve will serve to make the diagnosis. The x-ray will show a diseased bone.

GASTRIC CRISES.

Gastric crises have only to be remembered to be diagnosed. The Argyll-Robertson pupil and absence of the knee jerk will prove the presence of tabes to which the paroxysm is likely to be due. It must not be forgotten, however, that a patient may have both tabes dorsalis and gall-stones.

PANCREATITIS.

Pancreatitis, in its acute hemorrhagic state, is evidenced by marked pain in the epigastrium with collapse and symptoms of intestinal obstruction much more marked than are usual in gall-stones.

ANGINA PECTORIS.

Angina pectoris is frequently accompanied by pain having its chief point of severity in or about the gall-bladder region. The characteristic feature of the pain of angina pectoris is the fact that it is caused by excitement or exertion and is at once relieved by rest, either physical or mental. It may or may not be accompanied by cardiac distress or transference of pain to one or both arms.

PYLORIC OBSTRUCTION.

Pyloric obstruction, either organic or functional, may be accompanied by pain and vomiting; there is usually dilatation of the stomach. The gastric peristalsis can be observed. There is often the history of gastric ulcer. None of these symptoms are present in gall-stones.

Pericarditis and Pneumonia.

Pericarditis and pneumonia in the beginning may both simulate an acute gall-stone attack by reason of the fact that they are both accompanied by pain, the seat of which is sometimes transferred to the abdominal cavity. This area may correspond to either the appendiceal region or to the gall-bladder region. Attention to the condition of the heart, however, and of the lungs will show either or both of these organs the seat of inflammation. There is also the absence of reference of pain to the back, which is so common in gall-stones. They are both accompanied by leukocytosis, uncommon in simple gall-stone colic.

Acute Indigestion.

Acute indigestion certainly occurs, but it is accompanied by vomiting of undigested material; here, as in appendicitis, the vomiting and undigested food may be merely *symptoms* of the condition. Any acute indigestion which persists and which is accompanied by tenderness and leukocytosis is due to some organic cause.

Chronic Pancreatitis.

Chronic pancreatitis has pain as one of its symptoms together with marked disturbances of digestion. Examination of the stool for fat and undigested meat fibers and the absence of the characteristics of gall-bladder disease will be sufficient on which to at least base a diagnosis of pancreatitis.

Cancer of the Stomach and Bile Ducts.

Cancer of the stomach and of the bile ducts may have simple indigestion as one of its first and its most persistent symptoms. Here again, x-ray, continued examination of the stomach contents and stool must be the immediate steps toward a diagnosis. Operation must not be too long delayed. *The only treatment for cancer of the stomach which is valuable today is early operation.* Therefore, persistent examination of the stomach and the entire abdominal contents must be undertaken. These failing to give a negative diagnosis of cancer, an operation must be undertaken soon.

Chronic Results

Jaundice, as the result of gall-stones, may be fleeting or may last for weeks, months and sometimes for years. It increases and decreases in intensity according to the partial relief or continuance of the occlusion of the common duct.

Perforation of the gall-stone into neighboring viscera is common, even cutaneous fistulae have been found which discharge gall-stones. The symptoms of this are pain in the region of the gall-bladder, fever, leukocytosis, local tenderness and resistance; in other words, evidence of local inflammation together with marked collapse.

Inflammation of neighboring organs and adhesions binding the gall-bladder and ducts to neighboring organs are extremely common. They are indicated by interference of the function of these organs, by tenderness on pressure and by resistance.

Chronic cholecystitis or cholangitis is constantly mistaken for appendicitis, gastric ulcer, or lead colic. The best method of diagnosis from the first two is an exploratory incision; from the latter, the presence of a blue line and the degeneration of the red cells.

Obstruction of the bowels occurs when a stone which has entered into the gut is large enough to occlude the lumen of the gut, or where a kink has formed around it. This form of obstruction can rarely be diagnosticated unless there has been a set of symptoms connected with the gall-bladder, or unless some other evident cause of the obstruction is present. The symptoms of this obstruction are exactly like those of obstruction due to any other cause.

Intermittent fever with chills and increasing jaundice occur frequently when the stone is impacted at the ampulla of Vater. There is chill, high fever and sweat, followed in a few hours by increasing jaundice. This may simulate malarial fever, and is constantly mistaken for it. The history of the case with abdominal pain, etc., will easily classify the cases, but examination of the blood showing leukocytosis in the former and no leukocytosis and malarial organisms in the latter is proof positive.

The therapeutic test of the usefulness of quinin in ball valve stone and its specific action in malaria should be enough to conclude the diagnosis.

Chronic indigestion is constantly the diagnosis in the three conditions —gall-stones, appendicitis, and chronic gastric ulcer. In an individual who is proven to be free from alcoholism, from gross indiscretions of diet, from organic disease of the heart or kidney, if local examination and repeated examination of the feces and stomach contents is negative the case is at least not one of simple indigestion. We may be dealing with any of the above conditions or a beginning of carcinoma or chronic pancreatitis.

12. Cirrhosis of the Liver

Pathologists of recent times have discarded the terms of hypertrophic and atrophic cirrhosis, and for very good reasons. In certain stages of the so-called atrophic cirrhosis, or Lannec's cirrhosis, the liver is large instead of being small. The best terms perhaps are portal cirrhosis, which has reference to Lannec's cirrhosis, and Hanot's cirrhosis, or true biliary cirrhosis. Discussion of the differentiation of the cirrhoses will be limited to these two forms, remembering, however, that there are many other forms, such as syphilitic cirrhosis and cirrhosis due to perihepatitis, cirrhosis due to heart disease and local cirrhosis. These will not be discussed.

Portal Cirrhosis—Alcoholic Cirrhosis

Causes.—Portal, or alcoholic cirrhosis, is indicated first by digestive disturbances, which almost always occur as the result of the abuse of alcohol, or perhaps as frequently from overeating and lack of exercise. There are certainly some forms of true portal cirrhosis in which there is no history either of undue eating or of alcohol. Under these circumstances the most probable reason for such a condition is some intestinal intoxication.

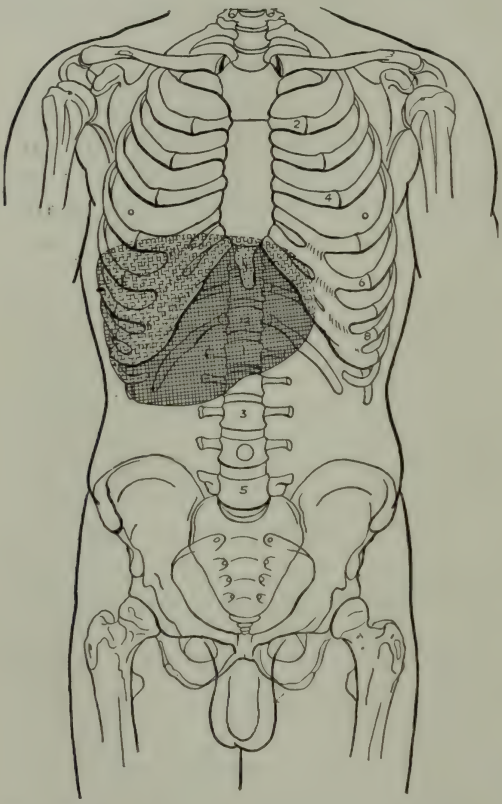


Fig. 57.—Area of Liver Dullness in Case of Portal Cirrhosis with Small Liver. Autopsy: The Liver was Tilted on Its Axis. (Original Observation.)

Symptoms.—In the first stages of portal cirrhosis, the liver is unquestionably larger than normal, and in an individual who is known to be a habitual drinker or who is known to be an overeater, an enlarged liver is always a suspicious indication of a beginning cirrhosis.

In the very beginning of some of the cases there are attacks of fever, accompanied by a feeling of weight in the hypochondrium or a decided indigestion, sometimes with slight jaundice; but these symptoms are not distinctive enough to make a positive diagnosis of cirrhosis. With the recurrence of the attacks of gastro-intestinal disturbances, the portal circulation begins to fail, and the next sign perhaps is enlargement of the spleen with enlargement of the abdominal vessels due to obstruction of the portal area.

Constipation is constant and tympanites is present. I repeat, that in these cases, these symptoms are not sufficient to make a diagnosis of cirrhosis: they are simply suggestive.

However, with the onset of the more severe contraction of the portal connective tissue, symptoms which are distinctive occur; among these hematemesis is the most positive. Frequently it occurs early in the case, sometimes it is the initial symptom. The writer has seen cases in which, except for a rather persistent gastritis, there have been no signs indicative of cirrhosis until a large vomiting of blood has occurred. This is due of course to large varices in the esophageal veins at the cardiac end. Hemorrhoids are common, due also to obstruction of the portal system. Then occurs dullness of the intellect, sometimes delirium, sometimes convulsions. Usually the condition is without fever, though attacks of fever occur at times.

The physical signs which make a diagnosis positive are ascites, with diminution or enlargement of the liver, enlargement of the abdominal veins and the formation of the caput medusæ about the umbilicus.

The writer has seen cases in which the liver was thought to be large, and which were proven at operation or autopsy to be a very small liver (Fig. 57) tilted on its axis. The distance between the beginning of the liver dullness in the chest and the edge of the liver felt in the abdomen was increased instead of diminished, so that the liver appeared large instead of small.

Conditions to be Differentiated from Cirrhosis of the Liver

This condition may be mistaken for:

Chronic indigestion due to other causes
Carcinoma of the liver
Carcinoma with cirrhosis
Chronic peritonitis
Retroperitoneal tumor
Failing heart
Duodenal or gastric ulcer
Banti's disease.

Perhaps the most important point is the recognition of the fact that true cirrhosis in the beginning has the symptoms of a simple gastritis. This is the time when prophylactic means are of value, and when abstinence from the use of alcohol will cause the condition to be arrested.

While one cannot make a positive diagnosis of cirrhosis in this early stage, it is such a well-known fact that alcohol is a common cause of cirrhosis, that a chronic indigestion occurring in an alcoholic is abundant excuse for exciting a fear of cirrhosis. Disappearance of indigestion, recession of the liver to its normal size, etc., will help to prove that no great degree of cirrhosis is present, although it is wise always to consider the condition a beginning cirrhosis, and to warn the patient.

CARCINOMA OF THE LIVER.

Carcinoma of the liver causes the rapid emaciation, with likelihood of decided jaundice and palpable masses appearing in the liver; these masses are often umbilicated. Ascites is somewhat less frequent in carcinoma than in cirrhosis.

CIRRHOSIS WITH CARCINOMA.

There is a form of cirrhosis with carcinoma, in which the diagnosis is absolutely impossible without an exploration. Here the individual has every sign of cirrhosis and also presents the rapid emaciation and jaundice which is so common in carcinoma.

CHRONIC PERITONITIS.

Chronic peritonitis may give rise to a collection of fluid in the abdomen which simulates ascites of cirrhosis, but there is abdominal pain; there is not as a rule the free transmission of the wave through the liquid in the abdomen. Tapping will usually show a purulent or bloody liquid in the abdomen.

A mass in the abdominal cavity may be mistaken for a liquid, but tapping or exploration will make the diagnosis certain. The liquid is likely to be bloody.

RETROPERITONEAL TUMOR.

A retroperitoneal mass may give rise to a true ascites due to pressure, but the removal of the liquid will reveal the mass.

FAILING HEART.

A failing heart will give rise to ascites, which might be mistaken for that due to cirrhosis; however, careful examination of the heart will show that organ to be the primary seat of the condition. On removal of the liquid there will be found an enlarged—not a small—liver.

DUODENAL OR GASTRIC ULCER.

Duodenal or gastric ulcer might be mistaken for or suspected to be cirrhosis in the presence of a gastric hemorrhage. Duodenal or gastric ulcer is usually relieved by eating and there are the intermittent attacks. Usually there is a history of more severe indigestion. The history of alcoholism will suggest cirrhosis.

Biliary Cirrhosis—Hanot's Cirrhosis

This is a disease of markedly chronic character. There is jaundice, enlarged liver and enlarged spleen, with frequent attacks of fever causing increase of jaundice. Ascites is rare.

This condition is extremely rare, but, curiously enough, the uninitiated are likely to consider any enlarged liver, especially if it is accompanied with jaundice, as due to Hanot's cirrhosis, this being probably due to the fact of lack of appreciation of the rarity of the disease, and the frequency of enlargement of the liver from other causes.

Conditions to be Differentiated from Biliary Cirrhosis

It may be mistaken for:

Portal cirrhosis
Obstructive jaundice
Carcinoma of the liver
Amyloid liver
Enlargement of liver due to cardiac decompensation
Enlargement due to syphilis, leukemia or malaria
Banti's disease.

PORTAL CIRRHOSIS.

In portal cirrhosis there is frequently a history of alcoholism; there is either a large or a small liver, usually the latter, though even in the late stage it may be large; small granular roughness can be felt over the surface of the liver in the late stage.

There is much ascites, and there is hematemesis, enlargement of the veins of the portal system; the spleen is moderately enlarged. There are clay-colored stools.

In biliary cirrhosis there is much enlargement of the liver; it is smooth. The spleen is greatly enlarged. There is no history of alcohol, no hematemesis and no ascites. There are attacks of pain and fever. The stools are not clay-colored, but contain bile pigment.

OBSTRUCTIVE JAUNDICE.

Obstructive jaundice might readily be mistaken for hypertrophic cirrhosis if only the recurrent attacks of fever and jaundice were taken into consideration. However, in obstructive jaundice, either the history of gall-stone attacks, attacks of biliary duct infection, a growth, or echinococcus can be obtained. The liver is not large, neither is the spleen; the stools are not colored with bile. There is frequently leukocytosis.

Carcinoma of the Liver.

Carcinoma of the liver is characterized by a history of indigestion, failing strength, the condition of the stomach contents often being indicative of carcinoma of the stomach. In carcinoma there is enlargement of the liver; often masses are felt over the liver surface; and jaundice is often present. The course is not so protracted as is that of hypertrophic cirrhosis.

Amyloid Liver.

In amyloid liver there is either the history of syphilis or of some long-standing suppurating disease. The patient is extremely anemic; there is frequently nephritis; there is no jaundice; the spleen is enlarged; there are no attacks of fever followed by jaundice. If there be fever it is from the original disease.

Enlargement of the Liver Due to Cardiac Decompensation.

There are all the signs of cardiac disease. The liver is enlarged, the spleen may be enlarged (it often is) but the case is not so chronic, as far as hepatic symptoms are concerned. There is edema of the legs. The attacks of fever followed by jaundice are conspicuously absent.

Enlargement Due to Syphilis, Leukemia, or Malaria.

These conditions rarely have jaundice as one of their prominent symptoms. They can be distinguished by the Wassermann reaction, leukemic blood and malarial organisms respectively.

Banti's Disease.

Banti's disease may also be mistaken for primary cirrhosis of the liver. However, the progressive anemia, the history of a large spleen antedating the history of signs of cirrhosis, will help to make the diagnosis. Indeed, Banti's disease is but a terminal portal cirrhosis in a case of splenic anemia.

13. Abscess of the Liver

Forms.—Abscess of the liver may be solitary or multiple. The first (*a*), *solitary* or tropical abscess, is due to infection by ameba coli, which is the causative factor in one form of tropical dysentery. The second (*b*), *multiple* or infective abscess, is due to streptococci or other infection from some area of the hepatic drainage field, such as the gall-bladder region, appendix, pancreas or splenic region.

These two forms because of their distinct symptomatology and differentiation will be considered separately.

(*a*) *Amebic Abscess*

Characteristic Features.—Amebic abscess almost without exception has the history of residence for a longer or shorter time in a tropical or subtropical territory, together with a dysentery—though the history of the latter may be vague. There is an intermittent fever, leukocytosis, pain and tenderness in the region of the liver, sallow complexion or a distinct jaundice. An x-ray picture shows a high domelike liver forming an acute angle with the spinal column as contrasted with the relative flat arch and oblique spinal angle of a normal liver. Ameba are found in the stools. Pus may be demonstrated by puncture of the enlarged liver. Unfortunately certain cases go unrecognized because of dearth of symptoms, but if after residence in a tropical region there is continued failure of health with enlarged liver, the diagnostician must at once bring all his resources to prove or disprove the presence of abscess. Among these *careful and repeated stool examination is most necessary.*

Conditions to be Differentiated from Amebic Abscess

This condition must be separated from:

Biliary cirrhosis or Hanot's cirrhosis
Carcinoma of the liver
Amyloid liver
Hydatid liver (See page 309).

BILIARY CIRRHOSIS OR HANOT'S CIRRHOSIS.

Biliary cirrhosis has a chronic course; there is jaundice and fever, but the latter comes on in attacks and is not continuous. The jaundice is a true discoloration of the skin and mucous membrane with bile, and is much more intense than the slight jaundice or sallowness of abscess. The spleen is enlarged; this is not the case in abscess; there is no leukocytosis, no history of dysentery and there is absence of ameba in the stools.

CARCINOMA OF THE LIVER.

Carcinoma of the liver usually has a shorter course. The enlargement of the liver is, in most cases, irregular and nodular, though it may be uniform and smooth in primary cancer; emaciation is more rapid; there are many more distinct stomach symptoms than in abscess; leukocytosis is less marked; there is usually more pain. Exploration will demonstrate a solid mass and not a collection of pus. There is no history of dysentery and no amebae.

AMYLOID LIVER.

In amyloid liver there is uniform enlargement; there is extreme anemia. Careful search will show the presence of either syphilis or some chronic suppurative process. If there is chronic suppuration, there may be leukocytosis; if there is syphilis, there will be a positive Wassermann reaction as indicative of the condition. The urine is very apt to show the presence of albumin, which condition is not indicative of abscess.

(*b*) *Multiple or Infective Abscess*

Characteristic Features.—Multiple or infective abscess is always secondary to some suppurative process in some other part of the body, and especially about the appendix, gall-bladder, spleen or pancreas.

The symptoms of acute appendicitis, of gall-bladder disease, or those of pancreatitis can usually be developed. It is acknowledged that the inflammatory conditions in these primary areas may be entirely overshadowed at the time of observation by the more urgent symptoms of fever, chills, high leukocytosis, jaundice and tenderness over the liver. The liver is not always enlarged. The abscesses may range from about microscopic size to that of a grape fruit, but are usually small and very numerous.

Conditions to be Differentiated from Multiple or Infective Abscess

The conditions with which it is confounded are:

Pneumonia
Subdiaphragmatic abscess
Supradiaphragmatic collection of liquid
Any one of the primary enlargements of the liver.

PNEUMONIA.

Pneumonia may simulate abscess by its sudden onset, chill, fever, leukocytosis and abdominal pain. The dullness of pneumonia may be simulated by a dullness over the right base in abscess, due to simple atelectasis, from lack of movement of the lung, or it may be due to an active congestion of the base of the lung adjacent to the affected liver. The condition may not be suspected, but the whole symptomatology be referred to the primary lesion, this being due to the dearth of symptoms referable to the liver.

Very soon the typical physical signs will show the case to be one of pneumonia. There is no history of a probable abscess or gall-bladder disease.

SUBDIAPHRAGMATIC ABSCESS.

In this condition there is the same history of infection in the region of the appendiceal or the gall-bladder region. The edge of the liver can be felt below the ribs; there is dullness far up in the chest; the movement of the right costal margin is less than the left. The x-ray and the fluoroscope will show the collection below the diaphragm.

HYDATIDS OF THE LIVER.

In this condition there is no leukocytosis unless the cyst suppurates; there is no pain; the enlargement is greater; tapping often shows hooklets of the echinococcus. Palpation of the mass will often give a curious friction fremitus which is characterized by a thrill felt below the hand.

SUPRADIAPHRAGMATIC COLLECTION OF LIQUID.

Suprаphrenic abscess, or a localized collection of liquid in the chest *above* the diaphragm, may be distinguished easily by x-ray and especially by the use of the fluoroscope. In suprаphrenic collections the diaphragm and liver may be seen to move independently of the shadow above. The lung may be very little compressed. However, there may be fixation of the diaphragm in certain of the conditions which will make the diagnosis more difficult. Here *incision* is about the only means of differentiation.

14. Cancer of the Liver

Forms.—Carcinoma of the liver is secondary or primary, the former being much more common.

Secondary Carcinoma.—Secondary carcinoma may follow cancer in any portion of the body, the stomach, rectum and uterus being the most usual sites of primary implantation. Enlargement of the liver, often small nodules over the liver, jaundice, loss of strength, rapid emaciation, sometimes ascites are the most prominent symptoms in a case which presents enough symptoms to make a diagnosis.

Primary Cancer.—Exactly the same symptoms occur in primary cancer, except that the enlargement is uniform, and umbilicated nodules are very rarely scattered over the liver.

Differentiation between the two forms can be made by the discovery of a primary focus in another portion of the body or the demonstration of distinct multiple growths in the liver itself. The latter, however, may be deceptive because the secondary growths occur in the liver substance, occasionally from the growth which is primary in the liver itself.

Conditions to be Differentiated from Cancer of the Liver

Differentiation is required from:

Syphilis
Cirrhosis
Amyloid liver
Sarcoma
Hydatid disease
Abscess of liver
Tumors of other organs
Fecal accumulations
Pleural effusion.

Syphilis of the Liver.

Syphilis of the liver is frequently mistaken for cancer. The enlargement here may be due to interstitial changes, or there may be nodular growths in the liver itself. The resemblance to carcinoma is most close; in carcinoma the history is not so prolonged as it usually is in syphilis, though the history of previous illness may be prolonged in carcinoma.

The Wassermann reaction is present in the luetic cases and absent in carcinoma, though of course carcinoma and syphilis may exist in the same patient. Above all, the use of antisyphilitic treatment is most prompt in the majority of cases of syphilitic enlargement of the liver. One must never forget that syphilis of the liver may be the underlying cause of almost any given enlargement, hence in cases in which a diagnosis cannot be certainly verified, a Wassermann test is always demanded, or a thorough trial of antisyphilitic treatment given.

Cirrhosis.—The cirrhoses are usually more chronic in form than is cancer. Both forms have enlarged spleen, both are more chronic in their course. In biliary cirrhosis there is a large smooth liver with intense jaundice and attacks of fever and depression, while in portal cirrhosis there is either a large tumor or a small rough liver with ascites, enlargement of the portal system of veins, and a tendency to hemorrhage from the esophagus.

Amyloid Liver.

Amyloid liver has a history of suppuration, usually albumin in the urine, and has a large smooth liver. There is no jaundice, as there may be with carcinoma.

Sarcoma.

The differentiation of sarcoma is largely one of pathology, though it must be remembered that sarcoma is common in childhood and may be mistaken for various other enlargements of the liver.

Hydatid Disease.

Hydatid cysts are rare. They may cause great enlargement of the liver. Frequently there is fluctuation and often there is a curious fremitus noticed in palpation. If the cyst is tapped, hooklets will be found.

Abscess of the Liver.

Abscess of the liver has the previous history of dysentery or some inflammation in the region drained by the vessels or lymphatics of the liver. There is fever, leukocytosis, often great pain over the liver region.

TUMORS OF OTHER ORGANS.

Tumors of other organs may be attached to the liver and move with it, but usually they can be moved independently of the liver and do not move with respiration. Careful attention to the signs and symptoms will usually make the diagnosis.

FECAL ACCUMULATIONS.

Fecal masses can be moved independently; they are painless; they can be indented; they can be removed by free catharsis.

PLEURAL EFFUSION.

Pleural effusion may be simulated by cancer of the liver in rare cases. The liver is so enlarged that the upper line of dullness may reach high in the chest. The heart may be pushed to the left. Examination by a needle will show either blood drawn from the liver or no exudate will be found. The accompanying figure is a cut of an x-ray in such a case (Fig. 58).

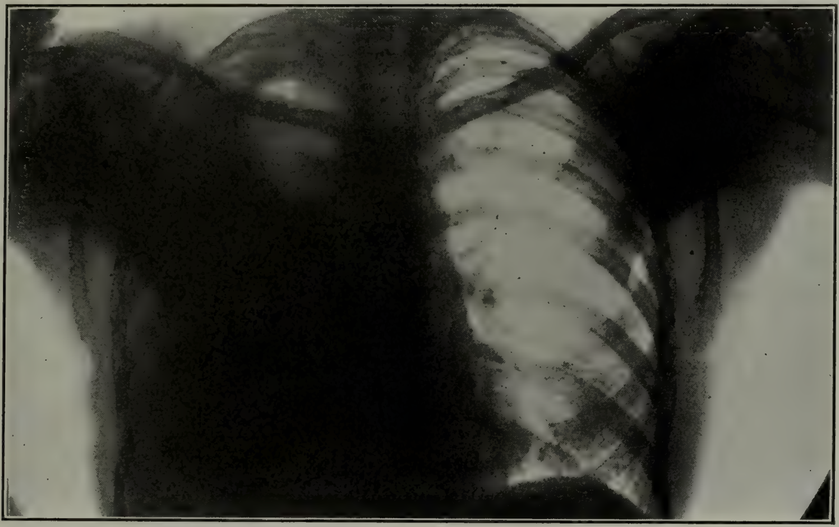


Fig. 58.—Cancer of Liver Simulating Pleural Effusion. Confirmed by Autopsy. (Original Observation.)

15. Fatty Liver

Origin.—A fatty infiltration of the liver is a frequent result of alcoholism or of obesity.

The symptoms are vague, and are due not to the fat contained in the liver substance, but either to disturbance of the function of the liver, to an accompanying cirrhosis or to the originating disease.

Symptoms.—The liver is enlarged and can be felt below the edge of the ribs, and is smooth.

Conditions to be Differentiated from Fatty Liver

It may be mistaken for:

Cirrhosis
Amyloid liver
Enlargement which accompanies cardiac decompensation
Enlargement due to leukemia
Cancer of the liver
Displaced liver.

CIRRHOSIS.

From cirrhosis it is distinguished by the dearth of symptoms. In cirrhosis all the symptoms—indigestion, vomiting of blood, edema, ascites, etc.—are present. In certain cases of fatty liver, however, there is a very decided cirrhotic change in addition to the fatty infiltration. Under these conditions it is impossible to determine the degree of fatty infiltration without an autopsy.

AMYLOID LIVER.

Amyloid liver is the result of prolonged suppuration due usually to syphilis or tuberculosis. The absence of these causes will argue absolutely against fatty degeneration.

ENLARGEMENT WHICH ACCOMPANIES CARDIAC DECOMPENSATION.

Enlargement due to passive congestion, the result of cardiac decompensation, may be diagnosed by the presence of this latter condition. There is not usually any fatty degeneration in such a liver unless the individual is an alcoholic.

ENLARGEMENT DUE TO LEUKEMIA.

Enlargement due to leukemia may be certainly diagnosed by the blood picture; indeed no differential diagnosis should be positively made without a differential blood count. Leukemia may be the cause of a cardiac failure and not recognized unless a count of the blood has been made.

CARCINOMA OF THE LIVER.

Carcinoma of the liver is accompanied by progressive loss of weight; the edge of the liver is not smooth as it is in fatty infiltration; carcinoma may be found in other portions of the body.

Displaced Liver.

A displaced liver can be felt below the ribs. If it is due to enteroptosis it is movable; if it is displaced by a collection, either above or below the diaphragm, the signs of that condition can be distinguished.

16. Suppurative Pylephlebitis

Etiology.—This condition is due to septic inflammation of the vessels of the liver, the infection having its origin in the terminals of the portal system such as vessels in the stomach, liver, mesentery, and appendiceal regions. It is also found in cirrhosis.

Diagnosis.—It is always difficult of diagnosis.

Suppurative phlebitis may be suspected when in any one of the above conditions there is a sudden onset of signs and symptoms of septic fever, increased leukocytosis, etc. It is particularly common in cases of neglected appendicitis. The writer has seen two cases of appendicitis in which operation was long delayed, one of which at the time of operation had multiple abscesses of the liver; the other made a remarkable temporary symptomatic recovery, to be followed in about two weeks with all the old symptoms of sepsis, and ended in death. At the autopsy the whole liver was found to be riddled with abscesses.

The condition, as stated, must always be suspected when a continued septic state follows an appendicitis (not necessarily suppurative in character) or inflammation in any of the region drained by the portal system.

Conditions to be Differentiated from Suppurative Pylephlebitis

It may be mistaken for:

A Local Suppurative Process Not Connected with the Liver, such as *perigastric abscess, subdiaphragmatic abscess, abscesses localized between coils of intestines,* etc.

The points of diagnosis are the occurrence of jaundice, enlargement of the liver and local tenderness in the hepatic region as distinguished from local signs elsewhere.

The use of an x-ray will often, but not always, distinguish between a subdiaphragmatic abscess and abscesses within the liver itself.

17. Amyloid Liver

This condition is characterized by enlarged smooth liver with few symptoms which have relation to the liver itself. It is the result of long-standing suppuration or of syphilis.

Conditions to be Differentiated from Amyloid Liver

Amyloid liver may be mistaken for:

Cirrhosis
Fatty liver
Leukemic infiltration
Carcinoma of the liver.

CIRRHOSIS.

It can be distinguished from cirrhosis by the fact that amyloid liver *per se* gives rise to very few symptoms. Amyloid liver is always the result of syphilis or long-standing suppuration—this fact makes the efficient differentiation.

FATTY LIVER.

In fatty liver there is a history of long-continued alcoholism; there is the flushed face with dilated vessels rather than the extreme anemia and marks of amyloid.

LEUKEMIC INFILTRATION.

From leukemic hepatic enlargement the condition is at once distinguished by an examination of the blood, showing the presence of a marked polymorphous leukocytosis.

CARCINOMA OF THE LIVER.

In this condition there is no history of suppuration or of syphilis, but there is often a history of carcinoma elsewhere in the body; there is rapid loss of weight; the liver may be nodular.

18. Anomalies of Size and Position of the Liver

Anomalies of size and position of the liver occur when there is enteroptosis. Rarely is the liver alone out of position, but almost without exception the stomach, intestines and spleen likewise have a lower position than normal.

Conditions to be Differentiated

They may be mistaken for:

Abdominal tumor
Enlarged liver
A liver pushed out of position.

A floating liver which is not enlarged may extend far into the abdominal cavity and tilt on its axis so that the upper margin may be percussed at or above the normal position, thus closely simulating an enlarged liver. This occurs often in cirrhosis of the liver. There is marked contraction of the organ; the lower edge can be felt far below the edge of the ribs, and the upper line of dullness observed at or near its normal position. This makes the case closely resemble one of cirrhosis with fatty infiltration. The confirmation of these facts is had either at operation or at autopsy. In Figure 57 the outline of such a liver is shown. At operation the liver was found to be extremely small; it was tilted on its axis and ptosed.

Abdominal Tumor.

An abdominal tumor, independent of the liver itself, is rarely the shape of the liver or in the region occupied by the ptosed liver.

A floating kidney or a large pyloric mass occupying this position might be thought to be a liver, but the shape would surely make the differentiation.

Enlarged Liver.

An enlarged liver is not movable except on respiration; there are usually symptoms which call attention to the condition. The examination gives the impression of a voluminous organ and not a merely small ptosed organ.

Liver Pushed Out of Position.

A liver pushed out of place by a collection either above the liver, below the diaphragm or in the chest above the diaphragm, can be diagnosed by the extension of the dullness upward and by signs of intercurrent disease.

A Riedel's lobe, or a portion of the liver connected with the main body by a fibrous band, a corset liver, will occasionally resemble a gall-bladder, a floating kidney, or a pyloric mass.

The diagnosis can be made by the fact that such a lobe usually moves with respiration, and gives no symptoms referable to the gall-bladder, kidney or pylorus.

I. Diseases of the Pancreas

The author is especially indebted for the literature and for much of the information in regard to the diseases of the pancreas to the work of Opie, in his monograph on "Diseases of the Pancreas," and in Osler's and McCrae's "System of Medicine."

1. Pancreatic Insufficiency

Some of the symptoms of pancreatic insufficiency are characteristic. The symptoms, according to Opie, depend upon whether the internal or external secretion is interfered with. When the external secretion is deficient there is disturbance of digestion, as the internal secretion has to do with the metabolism of carbohydrates.

Examination of the stools is necessary for a diagnosis. Large stools rich in fat and undigested meat fiber occur; there is flatulence, and the presence of Cammidge reaction speaks for a pancreatic insufficiency, though the distinct nature of the insufficiency may not be ascertained. This is shown by the presence of fat in the stool when the digestion of fat is interfered with, and the presence of undigested meat fibers when proteids are not well digested.

Steatorrhea.—The first, steatorrhea, is indicated by large stools of grayish color and of oily consistency. It is by no means always present when the pancreatic secretion is interfered with, but when such stools are present they indicate pancreatic insufficiency.

Azotorrhea.—The presence of protein in the stools, as indicated by undigested meat fibers and especially by the unchanged nuclei of muscle fibers, has been looked upon as especially valuable as an indication of interference with the pancreatic secretion.

Glycosuria.—Glycosuria and diabetes mellitus most frequently have to do with interference of pancreatic internal secretion.

Opie, in his "Diseases of the Pancreas," has shown that the vast majority of cases of glycosuria are accompanied by disease or atrophy of the islands of Langerhans. Though serious disease of the pancreas may occur without glycosuria, it is amply proven that in such cases enough of the islands of Langerhans remain to maintain the metabolism of carbohydrates.

Alimentary glycosuria may occur when there is no demonstrable lesion of the pancreas, but in these cases there is probably some functional interference with the islands of Langerhans.

2. Pancreatitis

Forms.—Pancreatitis can be divided into two forms, *chronic pancreatitis* due to irritation from gall-stones, pancreatic stones from intestinal irritation or from some toxic substance circulating in the blood; and *acute pancreatitis* due to acute hemorrhage or actual apoplexy of the pancreas itself, or to an ordinary suppuration of the pancreas, just as suppuration occurs in other portions of the body. A gangrenous pancreatitis usually follows hemorrhage into the pancreatic substance.

(*a*) *Chronic Pancreatitis*

Origin.—The first of these, chronic pancreatitis (interstitial pancreatitis) results in an increase in the connective tissue of the pancreas which gradually replaces the glandular substance itself.

Characteristic Features.—It is characterized by vague pains in the epigastrium, by indigestion, by flatulence, disturbance after eating—sometimes by sugar in the urine; it is occasionally accompanied by jaundice and also by the appearance of fat (steatorrhea) and meat fibers (azotorrhea) in the bowel movements.

Diagnosis.—Diagnosis of chronic pancreatitis is accomplished with considerable difficulty, the symptoms resembling closely those of gall-stones or stones in the pancreas itself. Very frequently the head of the pancreas is the part involved. The diagnosis, as a matter of fact, often depends upon the findings at operation.

According to Deaver, in an analysis of sixty cases of chronic pancreatitis, the symptoms are first disturbances of digestion and flatulence, followed by pain after eating. The relation of the pain to the taking of food is not constant. Nausea and vomiting are common; jaundice is present in two-thirds of the cases; constipation is the rule. Physical examination is of very little importance. Occasionally one may feel the pancreas lying across the abdomen, swollen and hard. Tenderness is of no value. Usually there is subnormal acidity of the stomach contents in simple pancreatitis, in cases complicated by gall-stones there is hyperacidity. Starch digestion is interfered with. Where the islands of Langerhans are interfered with sugar appears in the urine in the majority of cases, but where the sclerosis is interlobular there is no diabetes. According to some writers the Cammidge reaction is present in acute pancreatitis. In these cases examination of the feces is important: the stools are large, fatty, extremely bulky; undigested muscle fibers are present.

Conditions to be Differentiated from Chronic Pancreatitis

Chronic pancreatitis must be separated from:

Simple chronic indigestion
Gall-stones
Chronic appendicitis
Gastric ulcer.

By reference to the various chapters on the subjects of chronic indigestion, gall-stones, gastric ulcer and appendicitis, the points of distinction between these conditions and interstitial pancreatitis will be seen.

Simple Chronic Indigestion.

In chronic indigestion, which is so commonly dependent upon one of the various organic conditions affecting the stomach either directly or reflexly, one must be positive that organic conditions do not exist before the diagnosis of simple chronic indigestion is decided upon. Indeed the diagnosis is really only made by exclusion.

Gall-stones.

Pancreatitis is separated from gall-stones on the ground of less severe pain. The pain is less paroxysmal and does not radiate to the shoulders as is common in gall-stones, though a previous history of gall-stones is in favor of the existence of pancreatitis. The pain differs from that of gastric ulcer in that there is no direct relation of the pain and the taking of food.

Chronic Appendicitis—Gastric Ulcer.

In chronic appendicitis there is as a rule a spot of tenderness somewhere in the region of the appendix which is not present in gall-stones, gastric ulcer or pancreatitis. The stomach contents are usually subacid in pancreatitis, rather than hyperacid, as is frequently the case in gall-stones and in gastric ulcer.

The Cammidge reaction in the urine is absent. This urine examination has been found so unreliable and in need of so much accurate chemical knowledge that it must, to be of any value whatever, be made by an expert. The stools are *large, fatty,* characteristic white stools, containing, microscopically, much fat, often muscle fibers and starch granules. These stools are not common where the pancreas is not affected.

(*b*) *Acute Pancreatitis—Hemorrhagic Pancreatitis*

Hemorrhage into the substance of the pancreas may be of such slight amount that a diagnosis is impossible. It may follow instead of preceding a necrosis, which is found either at necropsy or at operation.

The form of this disease which is capable of diagnosis before death is characterized by *sudden,* severe abdominal pain referred to the epigastrium, with vomiting, marked collapse and anemia. The pain is continuous or intermittent. It is often the dominant symptom in the case and demands immediate attention, either by operation or by other means. There is constant vomiting; delirium often occurs with fever.

Diagnosis.—The diagnosis is always difficult before operation. Careful examination must be made for gall-stones, for a perforative peritonitis or for a gastric ulcer. One realizes, however, that he is in the presence of an apparently lethal disease. This condition is most difficult to differentiate from perforative peritonitis. In gall-stones, in gastric ulcer and frequently in appendicitis, the common causes of perforative peritonitis, there is usually the history of some antecedent distinctive disturbance which characterizes these various conditions. In hemorrhagic pancreatitis, on the other hand, the suddenness and severity of the onset is characteristic, and the pain is apt to be excruciating in character. Usually there is a leukocytosis, which is of no help in the diagnosis.

If the patient is not operated and survives for three or four days, the case is converted into what is usually designated the necrotic stage. Here the temperature takes on a typical septic aspect, ranging from 100° to 104° F., with increasing leukocytosis.

A tumor is likely to appear in the epigastrium due to collection of fluid in the lesser peritoneal cavity.

The diagnosis, from a postgastric collection due to a perforated gastric or duodenal ulcer, must depend absolutely on the previous history.

If there have been symptoms of these conditions there is likelihood of the abscess being secondary, but if the attack is sudden, severe, and apparently lethal, the condition is more likely to be due to a pancreatitis.

3. Calculus of the Pancreas

Calculus of the pancreas may be entirely without noteworthy symptoms, or it may have as its symptoms intermittent epigastric pains, without symptoms of jaundice, or the ordinary symptoms due to gastric ulcer. A tumor may result as the consequence of blocking of the pancreatic duct. The pancreatic fluid may be excluded from the intestine to such an extent that all the symptoms of pancreatic disease—wasting weakness, fatty stools and inability to digest meat, as shown by the recovery of meat fibers from the stools—are present.

During the passage of pancreatic calculi, chill and fever have been observed which closely resemble the attacks of Charcot's fever, with the important difference that they are not accompanied or followed by jaundice. Cases are reported, however, in which jaundice has been a temporary symptom when the calculus has passed into the ampulla of Vater. In a large number of the proven cases, glycosuria has been a symptom.

Conditions to be Differentiated from Calculus of the Pancreas

Biliary calculus
Gastric ulcer
Carcinoma of the stomach or biliary passages
Chronic pancreatitis
Cyst of the pancreas
Primary tumors of the pancreas.

Biliary Calculus.

Biliary calculi may have every characteristic of pancreatic calculi, or the reverse may be true. As a rule, however, persistent jaundice, intermittent fever with jaundice and persistent tenderness in the region of the gall-bladder indicate the condition to be due to gall-bladder disease. As Opie remarks, "the close study of all the proven cases of pancreatic calculi shows that in many instances an accurate diagnosis is impossible."

Gastric Ulcer.

Periodicity and chronicity of the attacks, relief of the pain after eating, together with blood in the gastric contents and stool, and hyperacidity are characteristic symptoms of gastric ulcer. These are all wanting in pancreatic calculi.

Carcinoma of the Stomach or Biliary Passages.

Carcinoma of the stomach may simulate the condition by the emaciation and pain. However, as a rule, blood is constantly in the stomach contents and stool of carcinoma, while blood is not present in the stool of pancreatic calculi. The characteristic stools of disease of the pancreas are absent.

Chronic Pancreatitis.

Chronic pancreatitis, without calculi, can only be diagnosed from calculi by less severe pain.

Cyst of the Pancreas.

A cyst of the pancreas may result from some other condition than a stone, in which case only the absence of a shadow of a stone will make the diagnosis.

Primary Tumors of the Pancreas.

A tumor of the pancreas, other than that resulting from blocking of a duct by a stone, may have the characteristics of a solid tumor; however, the differentiation is problematical.

4. Pancreatic Cysts

Pancreatic cysts are suspected by the presence of a cystic growth in the epigastrium.

Symptoms.—There is *pain,* varying both in degree and character; usually it is a deep-seated pain, and may occur in paroxysms resembling those of gastric crises.

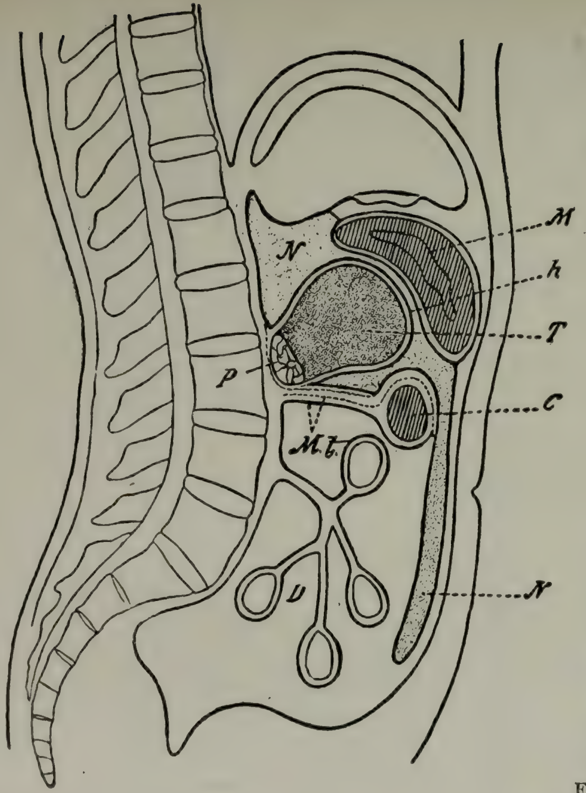


Fig. 59.—Tumor of the Ventral Surface of the Pancreas Projecting Into the Bursa. The Stomach Lies in Front of, the Colon Below, the Tumor. M, Stomach; C, Transverse Colon; P, Pancreas; D, Coils of Intestine; N, Omental Bursa; M.t., the Two Layers of the Transverse Mesocolon; h, Posterior Layer of the Great Omentum; T, Tumor. (After Osler and McCrae.)

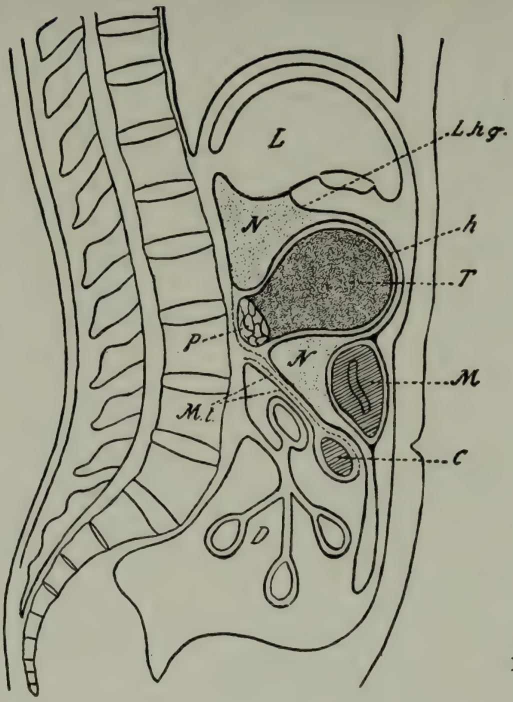


Fig. 60.—Tumor Projecting Into the Omental Bursa, Pushing the Lesser Omentum Forward. Stomach and Colon Lie Below the Tumor. L, Liver; M, Stomach; C, Transverse Colon; P, Pancreas; D, Coils of Intestine; N, Omental Bursa; L.h.g., Ligamentum Hepaticogastricum (Lesser Omentum); M.t., the Two Layers of the Transverse Mesocolon; h, Posterior Layer of the Great Omentum; T, Tumor. (After Osler and McCrae.)

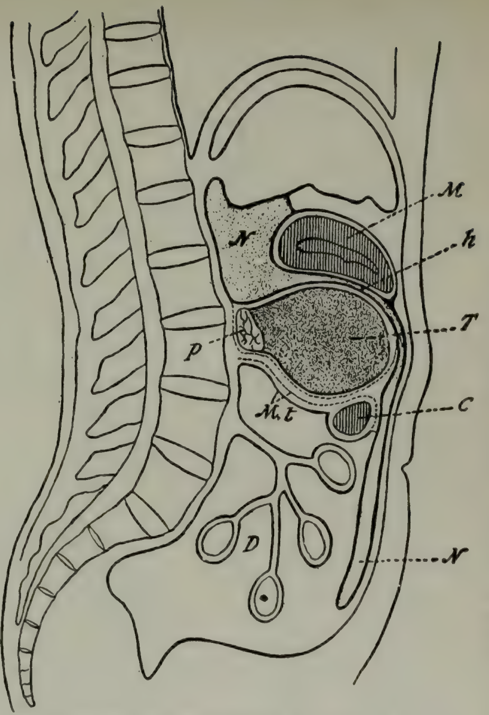


Fig. 61.—Tumor Symmetrically Developed in All Directions, Projecting Between the Posterior Layer of the Great Omentum and Transverse Mesocolon. Stomach Above, Colon Below, the Tumor. M, Stomach; C, Transverse Colon; P, Pancreas; D, Coils of Intestine; N, Omental Bursa; M.t., the Two Layers of the Transverse Mesocolon; h, Posterior Layer of the Great Omentum; T, Tumor. The Anterior Layer of the Great Omentum, which Is Pushed Forward Between the Stomach and Colon, Corresponds to the Gastro-colic Ligament. (After Osler and McCrae.)

Pressure Symptoms.—Gastric symptoms due to direct pressure are common. There are the symptoms of ordinary indigestion and no symptoms characteristic of a cyst *per se.* Jaundice due to pressure upon the common bile duct may make the condition inseperable from obstructive jaundice due to other causes. Ascites may result from pressure upon the portal vein, and edema of the feet and legs due to pressure upon the vena cava may result. Diabetes mellitus is sometimes present; loss of weight is common.

Physical Signs. — The physical signs are those of a cystic growth in the epigastrium. According to Korte, who publishes the accompanying figures, from "Modern Medicine," Osler and McCrae, the cyst may be present back of the stomach, above the stomach, between the stomach and the colon, back of the colon, and below the colon (See Figs. 59, 60, 61, 62 and 63).

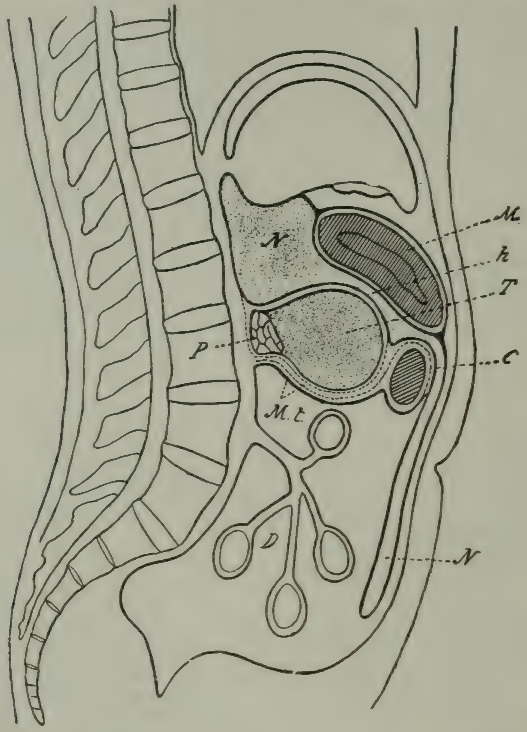


Fig. 62.—Tumor of the Same Region, Developed on One Side, Growth Largely Toward the Omental Bursa. Stomach Lies Above the Tumor, and Colon in Front of the Lower Portion. M, Stomach; C, Transverse Colon; P, Pancreas; D, Coils of Intestine; N, Omental Bursa; M.t., the Two Layers of the Transverse Mesocolon; h, Posterior Layer of the Great Omentum; T, Tumor. (After Osler and McCrae.)

Conditions to be Differentiated from Pancreatic Cysts

Retroperitoneal abscess due to traumatism or a ruptured gastric ulcer
Cancer of the pancreas
Retroperitoneal sarcoma
Cysts of neighboring organs
Aneurism of abdominal aorta.

Retroperitoneal Abscess.

Retroperitoneal abscess has the history of a traumatism or of a gastric ulcer preceding the appearance of the cyst; there is leukocytosis and much tenderness over the cyst. These are not common in a retention or proliferation cyst.

CANCER OF THE PANCREAS.

Carcinoma of the pancreas is differentiated by the fact that there is emaciation and probably jaundice. The tumor is solid rather than cystic.

RETROPERITONEAL SARCOMA.

Retroperitoneal sarcoma is solid, is not fluctuating, has no jaundice, and is likely to be quite fixed.

CYSTS OF NEIGHBORING ORGANS.

Cysts of other organs, the liver, the gall-bladder, the mesentery and the kidney must be differentiated by special signs present in these organs.

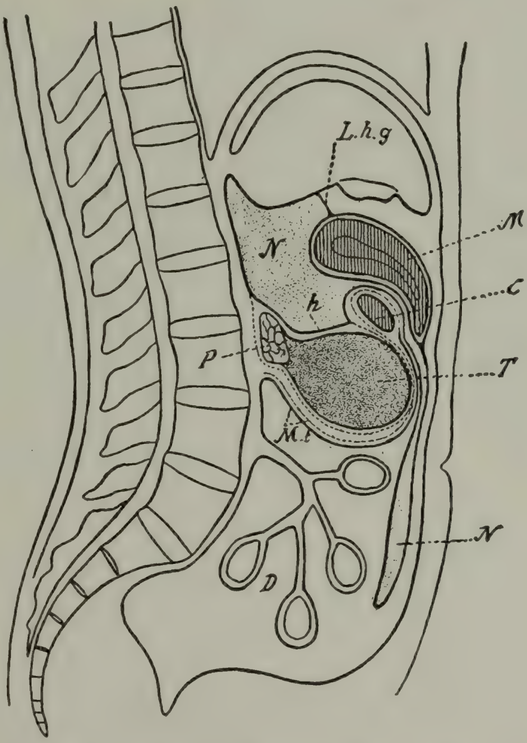


Fig. 63.—Tumor of the Same Region Developed on One Side, Growth Largely Downward Toward the Mid-abdomen. Tumor Lies Below the Colon and Stomach. M; Stomach; C, Transverse Colon; P, Pancreas; D, Coils of Intestine; N', Omental Bursa; L.h.g., Gastrohepatic Ligament; M.t., the Two Layers of the Transverse Mesocolon; h, Posterior Layer of the Great Omentum; T, Tumor. (After Osler and McCrae.)

An echinococcus cyst may simulate exactly a pancreatic cyst, which is between the liver and the stomach, but if the cyst is between the stomach and the colon, it is almost surely not connected with the liver. A puncture of the cyst will show the hooklets belonging to the worm. Occasionally one can discover the friction fremitus characteristic of echinococcus cyst. The cyst also may move with the liver, as does a cyst of the gall-bladder, unless the neck of the gall-bladder be unusually lengthened.

A cyst of the mesentery is usually movable.

Ovarian cysts are as a rule easily distinguished by signs and symptoms showing them to originate in the pelvis and not in the upper part of the abdomen.

A hydronephrosis may be so large that it occupies the entire abdominal cavity, but usually there is no difficulty in demonstrating the lumbar origin of the tumor. The origin of a pancreatic cyst is always central. Here an x-ray picture will be of much help, as will also catheterization of the ureters.

ANEURISM OF ABDOMINAL AORTA.

An aneurism of the abdominal aorta pulsates, and the pulsation is always expansile, while pulsation in a pancreatic cyst is always transmitted. A fluoroscopic examination will be of the greatest value.

5. Tumors of the Pancreas

Malignant disease and benign tumors both exist. The varieties are carcinoma, sarcoma and adenoma. Cysts have previously been described.

Carcinoma may be either primary or secondary.

Adenomata rarely give symptoms.

Carcinoma of the pancreas gives symptoms which have much to do with its position and size. When it is situated in the head of the pancreas there is almost always jaundice. Until the growth affects the general condition jaundice in small tumors of the head of the pancreas is often the sole symptom. Diabetes occurs if there is destruction of the islands of Langerhans. Pressure symptoms, disturbance of digestion, pain and epigastric tumor, with fatty stools and stools containing undigested meat fibers, often occur; cachexia and wasting eventually follow. Jaundice due to carcinoma of the pancreas is frequently accompanied by enlarged gall-bladder. Courvoisier's "law" states that jaundice with contraction of the gall-bladder points to gall-stones; jaundice plus dilatation of the gall-bladder indicates obstruction from other causes.

Conditions to be Differentiated from Tumors of the Pancreas

The condition can be confused with:

Gall-stones
Carcinoma of other organs
Intestinal obstructive symptoms.

GALL-STONES.

Gall-stones are distinguished by the intermittent pains, jaundice coming on in paroxysms, tenderness over the gall-bladder, often fever and leukocytosis. While jaundice may increase and continue for many years as the result of gall-stones, cachexia and loss of weight do not result—at least to any such degree as with carcinoma. There are not the stool changes which occur in carcinoma.

CARCINOMA OF OTHER ORGANS.

Tumors from other organs are usually accompanied by symptoms referable to the special organs. They are more movable than carcinoma of the pancreas.

Carcinoma of the pylorus may cause dilatation of the stomach, and while carcinoma of the head of the pancreas may cause dilatation of the stomach by pressure it is much less likely to cause dilatation than is the pyloric tumor.

Carcinoma of the colon may be simulated by pressure of the pancreatic tumor, causing an obstruction of the interior of the colon; but this is rare.

INTESTINAL OBSTRUCTIVE SYMPTOMS.

Intestinal obstructive symptoms may result from pressure or formation of adhesions because of the pancreatic tumor. A tumor mass will help to make the diagnosis.

J. Diseases of the Peritoneum

1. Peritonitis

Peritonitis may be diffuse or local. It may be the result of traumatism, of sepsis, rupture of viscus, or it may be toxic.

Local peritonitis is best exemplified in attacks of inflammation around the vermiform appendix which tend to recovery. Inflammation around the seat of an old gastric ulcer, about the gall-bladder and gall-ducts and about the spleen, liver, uterus and adnexa—all are excellent examples of local peritonitis. These attacks of local peritonitis are always the result of some inflammatory condition extending from the organ involved to the surrounding peritoneum.

Characteristic Features.—They are characterized by severe pain—local in character—depending upon the situation of the inflammation, local tenderness, usually slight fever and local resistance, together with a leukocytosis.

Conditions to be Differentiated from Peritonitis

FUNCTIONAL DISORDERS.

These attacks are constantly mistaken for some functional disorders or for some neuralgic or rheumatic condition. They must be distinguished from these; they must also be distinguished from spinal caries and neuritis affecting the lumbar and lower thoracic nerves, and from a local inflammation of the abdominal wall.

The fact that there is fever and severe pain which are made worse by motion of the body, local tenderness, local resistance and leukocytosis, at once removes the thought of a simple functional disorder. We should reserve the term "cramps," intestinal colic, and so forth, for conditions which we are positively sure are not the result of a local inflammation.

SPINAL CARIES.

When the lumbar or lower thoracic vertebrae are affected, there is frequently accompanying tenderness of the abdominal wall with resistance over the painful area and some fever. The only possible method of diagnosis is the examination of the spinal column for limitation of motion and for tenderness along the spine in every case. In spinal caries there is practically always tenderness and local limitation of motion. It does not exist in local peritonitis. The x-ray will show rarefaction or destruction of the vertebrae.

NEURITIS.

Neuritis affecting the lower thoracic or lumbar nerves is characterized by local tenderness and by extreme pain. This tenderness and pain, however, are usually along the entire length of the nerve from the spinal column to the anterior extremity of the nerves. This neuritis is often accompanied by herpes, the so-called herpes zoster. The tenderness is very superficial and there is rarely local resistance.

(*a*) ***Diffuse Peritonitis***

This is a much more serious condition. In the great majority of cases it is a sequel to some inflammation of an underlying organ.

Characteristic Symptoms.

1. *Pain.*—This may be sudden and severe as in perforation of a viscus, or it may develop gradually as in some cases of tubercular peritonitis, or it may be unnoticed by the patient on account of the toxemia existing.
2. *Collapse* occurs in cases of perforation of a viscus. This symptom is absent when the inflammation is due to tuberculosis or to some systemic poison.
3. *Abdominal rigidity* and contraction are common as the first symptoms of acute diffuse peritonitis. This is followed by rapid distention of the whole abdomen, often so great that the liver is pushed upwards. The patient lies with the legs drawn up—a most characteristic posture.
4. *Vomiting* is common; in the later stages there is regurgitation of dark material. The face is pinched; the pulse is quick and wiry; there is fever varying in intensity.

Conditions to be Differentiated from Diffuse Peritonitis

Intestinal obstruction
Acute dilatation of the stomach
Atony of the intestine
Lead colic
Hysteria
Pleurisy and pneumonia
Acute pancreatitis
Abdominal hemorrhage
Tympanites
Uremia.

Intestinal Obstruction.

This condition may be sudden or gradual in its onset. There is no fever, and no leukocytosis. Early in the course of the disease there is active peristalsis; there is absolute constipation; the coils of the intestine can be seen moving beneath the skin; the vomiting soon becomes fecal in character; frequently there is a history of previous abdominal operations, or a hernia is present. In children there is often a bloody stool as one of the first indications. The above symptoms occurring in aged individuals are very suspicious signs of sudden obstruction following partial obstruction due to a new growth.

Acute Dilatation of the Stomach.

Acute gastric dilatation is accompanied by great abdominal distention. The distention may be shown to be gastric both by percussion and by a succussion splash over the stomach. There is marked collapse, obstinate constipation, and the vomitus is large in amount and of putrid odor, although not truly fecal. A stomach tube can be passed, which will relieve the greatly distended stomach.

Atony of the Intestine.

Intestinal atony is chronic in character. There is no pain; bowel movements are normal but delayed; there is emaciation.

Lead Colic.

Lead colic, by reason of the abdominal pain, may be mistaken for peritonitis, but the history of the case, the blue line upon the gums and the degeneration of the red blood cells will make the diagnosis positive.

Hysteria.

A history of hysteria in the patient, the presence of abdominal distention with tenderness on pressure, the superficial tenderness being as great as that elicited by deep pressure, the soft character of the pulse, the absence of fever and leukocytosis and the general appearance of the patient, will put the case in the category of hysteria.

Pleurisy and Pneumonia.

Here there is the presence of a friction in the chest, there is dullness on percussion, and in pneumonia, signs of consolidation of the lung.

Acute Pancreatitis.

In this condition there is collapse, often great tenderness over the epigastrium and, at times, a large tumor lying crosswise of the abdomen.

Abdominal Hemorrhage.

In abdominal hemorrhage abdominal pain, leukocytosis and collapse are symptoms. There is usually dullness in the flanks and rapidly occurring anemia.

Tympanites.

Tympanites can be distinguished from peritonitis by the absence of tenderness, by the lack of leukocytosis, and the absence of fever not attributable to the general disease.

Uremia.

In certain cases of uremia abdominal pain and distention are among the symptoms. Examination of the urine, high blood pressure, and hypertrophied heart, will help in the diagnosis.

(*b*) *Perforating Peritonitis*

Perforating peritonitis is characterized by suddenness of onset, abdominal contraction followed by distention, obstinate constipation, frequent vomiting of large quantities of greenish black liquid, extreme pain which literally demands morphia for its relief, and by the absence of peristalsis. There may or there may not be a leukocytosis, depending upon the grade of inflammation and resistance of the patient. It is always septic in character.

(*c*) *Septic Peritonitis*

Septic peritonitis is the result of inflammation over a local abscess in some portion of the abdominal cavity, such as about the appendix, fallopian tubes or bile ducts. There are general symptoms after its onset not to be separated from perforative peritonitis.

Conditions to be Differentiated from Septic Peritonitis

Perforating Peritonitis cannot be distinguished from septic peritonitis.

The Peritonitis which Accompanies Puerperal Septicemia is almost always the result of extensive inflammation around the uterine adnexa.

(*d*) *Chronic Peritonitis*

Diffuse chronic peritonitis may be tuberculous, malignant, or the result of some inflammation which has become quiescent but has left behind many adhesions and consequently more or less interference with the intestinal functions.

The *tubercular form* has been described.

The *malignant form* is characterized frequently by the onset of pain, abdominal distention and more or less constipation. It cannot be diagnosed without the knowledge of malignant disease. With the knowledge of the presence of a malignant tumor, diffuse abdominal pain, distention, with more or less accumulation of liquid in the abdominal cavity, and with resistance, a diagnosis can be made. The liquid can, of course, be discovered by the ordinary methods of abdominal examinations. In either form there may be an abdominal mass due to an inflamed and rolled up peritoneum.

Conditions to be Differentiated from Chronic Peritonitis

The conditions with which this can be confused are:

Abdominal distention due to intestinal atony or incomplete obstruction
A collection of fluid free in the abdomen
Abdominal tumor.

ABDOMINAL DISTENTION DUE TO INTESTINAL ATONY.

Intestinal distention due to atony is usually painless; percussion over the entire abdomen gives a tympanitic note. No liquid can be discovered in the flanks. There is no history of tuberculosis, malignancy, or previous inflammation.

ABDOMINAL DISTENTION DUE TO INCOMPLETE OBSTRUCTION.

Intestinal obstruction (incomplete) is characterized by obstinate constipation, small stools, great abdominal distention, visible peristalsis and pain, the latter somewhat relieved by a bowel movement. There is no dullness; peristalsis is evident.

COLLECTION OF FREE FLUID.

Liquid from other causes in the abdomen can be detected by dullness in the flanks with a succussion wave. Often, as before stated, there is liquid with peritonitis; the simple ascitic liquid is free from pain or friction rub. There are symptoms of disease of the liver, or of cardiac decompensation which are wanting in true chronic peritonitis.

ABDOMINAL TUMOR.

A large cystic ovarian tumor may give rise to a succussion wave, but there is tympany in the flanks with dullness in the midabdomen. Frequently vaginal examination will show the presence of a tumor. An ovarian cyst is almost always multilocular; the loculi filled with liquid might be mistaken for walled-off collections of fluid.

2. New Growths of the Peritoneum

These may be either malignant masses or tubercular masses, which are difficult to differentiate. Benign tumors and cysts also occur.

Tubercular peritonitis has been described.

Malignant disease of the peritoneum, usually carcinomatous, has the symptoms of a chronic peritonitis—pain, distention of the abdominal cavity and ascites, plus rapid loss of weight and strength. When the ascitic fluid is withdrawn it is likely to be bloody or of a milky color, the last resembling a chylous fluid, or it may be entirely clear. Examination of the liquid often shows large polymorphonuclear cells. There are apt to be enlarged glands, both inguinal and axillary. The growths in the peritoneum are almost without exception secondary to malignant growths in other portions of the body.

Conditions to be Differentiated from New Growths of the Peritoneum

Portal cirrhosis
Ascites due to pressure or cardiac decompensation
Tumors of the mesentery
Non-malignant tumors
Hydatid cysts
Retroperitoneal tumors
Tumors in other organs.

PORTAL CIRRHOSIS.

Portal cirrhosis frequently has alcoholism as an antecedent. There is no primary malignant growth. When the ascitic fluid is withdrawn it does not reveal any new growths in the abdominal cavity; the liquid is clear and does not contain polymorphonuclear cells; the granular liver and enlarged spleen can often be felt after removal of the liquid.

ASCITES DUE TO PRESSURE OR TO CARDIAC DECOMPENSATION.

The last will have the accompanying and unmistakable signs of cardiac failure. A tumor giving pressure can be observed on removal of the ascitic fluid. The fluid itself is neither bloody nor cloudy and does not contain unusual cells.

Tumors of the Mesentery.

Tumors of the mesentery are likely to be movable; they are frequently secondary.

Non-malignant Tumors.

Benign tumors of the peritoneum itself can only be differentiated by withdrawing the liquid and discovering the tumor. Its true character can only be discovered by operation or autopsy.

Hydatid Cysts.

These occur very seldom and are constantly mistaken for ovarian cysts; they give rise to pressure symptoms. Their cystic character distinguishes them from malignant growths. If eosinophilia occurs it suggests echinococci; tapping will reveal hooklets.

In these days of progressive surgery the diagnosis of a cyst is sufficient reason for an operation which will serve at once to distinguish the condition from an ovarian cyst.

Retroperitoneal Tumors.

These masses are hard, not movable, and can be discovered to be independent of any of the viscera. When ascites occurs with them they are often disguised by the presence of the liquid. Removal of the liquid will show a hard immobile mass lying along the center line of the body.

Tumors of Other Organs.

Tumors of other organs can be recognized by their position and often by disturbances of the functions of the organs involved.

3. Ascites

Definition.—Ascites is a freely movable collection of liquid in the abdominal cavity.

Causes.—This results from some interference with the portal circulation or from some pressure on the abdominal vessel by a growth, or as the result of a chronic peritonitis.

Symptoms.—The symptoms are distention of the abdomen with dullness in the flanks. A succession wave is elicited by an assistant making firm pressure over the center line of the abdomen with the edge of the hand, while the observer places the palm of the hand on one flank and gently taps the flank on the opposite side; the wave is transmitted through the liquid to the opposite side. There is tympany over the region of the upper abdomen; the umbilicus protrudes. Occasionally there is resonance in the flanks, and at times there is a ring of dilated veins around the umbilicus—caput medusæ.

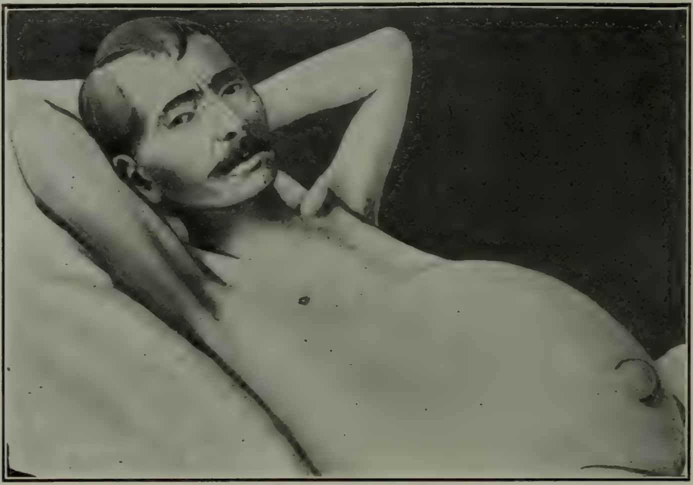


Fig. 64.—Distention of Abdomen Due to Ascites. (From Nothnagel's System, Published by Saunders.)

Conditions to be Differentiated from Ascites

Ovarian cyst
Distended bladder
Obesity of the abdomen
Dilatation of the stomach
Cysts of abdominal organs
Pregnancy.

Ovarian Cyst.

Ovarian cyst is likely to be situated to one side of the abdomen, but if very large it may occupy the central part. There is *dullness in the upper part* of the abdomen and tympany in the flanks; there may be the succussion wave through the mass of liquid as in ascites; frequently the cyst is lobulated.

Distended Bladder.

A distended bladder may be mistaken for ascites and has been tapped for such. The same physical signs exist in the abdomen as in the case of ovarian cyst—dullness over the tumor with resonance in the flank. Where there is the slightest doubt, catheterization should be performed, which will at once make the diagnosis positive.

Obesity of the Abdomen.

Abdominal fat has been mistaken for ascites. The diagnosis is not as simple as it would seem, especially where fluid and a fat abdomen coexist. A deceptive wave may be felt through a very fat abdomen, especially if there is insufficient pressure made in the median line; however, if sufficient pressure is made, no wave will be felt. There is no dullness in the flanks, though the percussion note approaches that if the abdomen is very pendulous. The belly wall can be pinched between the two hands, when it can easily be seen that the distention is not due to a liquid.

Dilatation of the Stomach.

In gaseous abdominal distention there is no dullness in any portion of the abdomen; there is no succussion wave. Dilatation of the stomach or intestines might be mistaken for ascites when the greatly dilated stomach or intestines contain much liquid. Here a splash heard makes a positive diagnosis of liquid plus gas. This can practically be only within the stomach or intestines. The stomach can be outlined, and a stomach tube will prove the presence or absence of liquid within the stomach.

Cysts of Abdominal Organs.

Other conditions such as *encysted liquid* due to peritonitis, a *cystic kidney, pancreatic cyst,* or a *large cyst of the liver* are to be differentiated. However, as ascites is almost without exception due either to disease of the heart or of the liver, the absence of these conditions will help to make the diagnosis. Most of the above conditions are unilateral and do not occupy the entire abdomen.

Cystic kidney very rarely occupies both sides of the abdomen. The history is one of long standing; there is no disease of the liver or heart.

In *peritonitis,* with a localized collection of fluid, there is a history of an attack of peritonitis; the encysted liquid rarely fills the entire abdomen.

Pregnancy.

Pregnancy with a large collection of amniotic fluid might be mistaken for ascites, but there is the usual history of pregnancy; careful vaginal examination will help to make the diagnosis. An x-ray may be used to demonstrate the fetal bones.

A new growth may cause such a combination of distention, solid material and liquid, that a diagnosis may be very difficult—but a new growth never causes the succussion wave, unless the growth is small and the amount of liquid enormous.

Section VI

Diseases of the Respiratory Organs

A. Diseases of the Nose

1. Coryza

Acute coryza is characterized by an inflammation of the nasal mucous membrane which results in sneezing—a flow of clear mucus followed by mucopurulent material. There is always lacrimation. Usually the condition is a part of the symptomatology of the ordinary "cold."

Conditions to be Differentiated from Coryza

This form may be confounded with:
Hay-fever
Coryza due to iodism.

HAY-FEVER.

Hay-fever may be differentiated by the fact that it appears at certain seasons of the year, the individual having been frequently affected at the same season. Usually there is not the same general depression; there is no fever. If often occurs in paroxysms appearing suddenly and on certain dates.

CORYZA DUE TO IODISM.

The best way of differentiating a coryza due to potassium iodin is to elicit the history of the use of the drug. Therefore a sudden coryza should always lead to an inquiry regarding the taking of potassium iodid.

There is often the same general distress of the limbs which accompanies a general infectious cold, but there is no fever and the salivary glands are much more likely to be involved.

2. Membranous Rhinitis

Membranous rhinitis is characterized by the formation of a membrane in the nose. It is frequently diphtheritic in origin. The symptoms are those of an acute rhinitis, but examination of the nares will show the presence of a membrane. Examination must be made of this for Klebs-Löffler bacilli; it is only by the absence of this bacillus that a simple rhinitis can be distinguished from diphtheria.

3. Epistaxis

Causes.—Bleeding from the nose may be the result of a blow, of local disease of the nose, or of an infection; it may be a part of the symptomatology of purpura or a hemolysis from any cause.

Diagnosis.—It can be mistaken for scarcely any other condition, though a careless observer might conclude that the blood which flows from the nose as a result of a large pulmonary hemorrhage or vomiting of blood was due to epistaxis alone. There is great danger of mistaking a pulmonary hemorrhage for a nose bleed or bleeding from the throat. It is safe to consider all spitting of blood as a pulmonary hemorrhage until proof to the contrary is furnished.

Epistaxis may be certainly diagnosed when a bleeding point is seen in the nasal cavities and when the pharynx, lungs and stomach are proven *not* to be the source of the blood.

B. Diseases of the Larynx

1. Laryngitis

Laryngitis may be acute or chronic.

Etiology.—It may be catarrhal, due to infection which gives rise to ordinary colds the exact nature of which is not known, or it may be due to almost any of the specific organisms such as tubercle bacilli, the spirochete of syphilis and to diphtheria bacillus. It may also be due to a general disease, nephritis. Occasionally there is a true spasmodic condition due to local inflammation.

Characteristic Features.—The disease, laryngitis, is characterized by cough, hoarseness and a rather dry spasmodic cough whatever the cause. It may be diagnosed symptomatically by the fact that there is hoarseness, more or less pain over the larynx and by the fact that the cough is aggravated by talking.

Diagnosis.—A positive diagnosis can be made by examination of the larynx with a mirror—an examination that all practitioners should be able to perform.

The forms of inflammation due to syphilis and tuberculosis, almost without exception give rise to ulceration of greater or less extent. In the beginning there may be mere infiltration and swelling of the parts. There are some characteristics which distinguish the two varieties, ulceration being common to both. Birkett, in Osler and McCrae's "Modern Medicine," gives the following points of difference:

> "Symptoms of Syphilitic Laryngitis: The most common lesions of the secondary stage are: first, erythema; secondly, superficial ulceration; thirdly, a mucous patch; and fourth, condylomata. Upon laryngoscopic examination the mucous membrane will be found either to be uniformly hyperemic, presenting essentially the same appearance as that of an ordinary acute laryngitis, or it may show an irregularity in the distribution of the inflammatory areas, this being due to interposed areas which are non-vascular, and the whole picture presenting a so-called 'mottled' appearance, which as some authors maintain, is definitely characteristic of secondary syphilis. The areas involved are generally the epiglottis and the false and true cords. This inflammatory process may lead to a destruction of the superficial layer of the mucous membrane, in which case there will be seen a small shallow and irregularly shaped ulcer whose surface is covered with a yellowish colored secretion. The superficial ulcers may extend and unite with others and when healed leave a very thin, stellate-looking cicatrix. The occurrence of the mucous patch within the larynx is comparatively rare. The laryngeal patch is rounded, oval or oblong in outline, of whitish gray or yellowish color, and surrounded by an area which is very hyperemic. The localities in which such a patch may be seen are the laryngeal surface of the epiglottis and its edges, the aryteno-epiglottidean fold, the false and true cords. Condylomata in the larynx appear as rounded or oval elevations with a yellowish colored surface.
>
> "In tuberculosis the ulcers are apt to be numerous, the outline not so sharp or distinct, the edges less indurated, the surface not so deeply excavated, and the granulations pale and indolent looking. The mucous membrane of the soft palate, pharynx, and larynx is pale; there is some febrile disturbance, with increased rate of pulse, and the general appearance of the patient is that of anemia. Smears from the ulcerated area will often show tubercle bacilli, and an examination of the expectoration will generally give a like result. The two diseases may coexist and an ulceration originally syphilitic may become tuberculous."

The ulceration may be so severe that locally it is impossible to distinguish these two forms of laryngitis; therefore either a physical examination revealing tuberculosis or a Wassermann complement fixation showing syphilis must be depended upon to differentiate. If the two conditions coexist diagnosis must depend entirely upon the finding of the organism of the particular condition in the lesions themselves.

The various forms of laryngitis must be differentiated from:

Laryngismus stridulus, or false croup, the result of rickets or tetany
Spasmodic catarrhal laryngitis
Diphtheritic laryngitis
Edema of larynx
Aneurism of the arch of the aorta
Enlarged thymus gland.

LARYNGISMUS STRIDULUS.

In laryngismus stridulus the condition comes on at any time of the day. It is chronic in character; there is no sign of an acute infection either catarrhal or diphtheritic; the child is usually rachitic or may have the characteristic tetany of the extremities. There are often tetanic spasms accompanying the throat affections.

SPASMODIC CATARRHAL LARYNGITIS.

Spasmodic laryngitis usually begins suddenly during the night—between midnight and 2 a.m. There may have been slight hoarseness before, but the child is suddenly awakened by a hoarse, croupy cough and difficult inspiration. Often he struggles for breath, becomes cyanosed and appears to the alarmed parents to be at the point of death. Usually a simple emetic such as ipecac or the inhalation of steam will allay the paroxysm, and the child will fall into a sleep. Examination of the throat *does not show* any white exudate in any position. When the child cries the note is loud and high pitched, and is not smothered.

DIPHTHERITIC LARYNGITIS.

Diphtheritic laryngitis is usually preceded by a gradually increasing hoarseness during the day; toward evening inspiration and expiration become more difficult, the breathing much more impeded. Sometimes there is a sudden increased difficulty of breathing, but usually the progress is gradually toward obstruction; the child cannot cry aloud, the cry being smothered. If asked to make a high note (*A* for instance) he cannot do so, while a child with spasmodic laryngitis can usually but not always reach the note. Examination of the throat will frequently reveal a white spot of membrane somewhere in the throat.

The breathing may be relieved by inhalations of steam or by emetics, but the distress quickly returns. The least appearance of exudate upon the larynx is indication sufficient to make a tentative diagnosis of diphtheritic croup.

Laryngeal examination which is difficult and often impracticable at the first visit will show the presence of membrane on the cords or in the larynx. In any case which gives reasonable doubt of its character, a large dose of antitoxin should be administered *at once.*

In certain cases a foreign body may give rise to signs of acute laryngitis; the history of inspiration of some object as a small bit of food must be carefully inquired into, and an examination of the larynx be made.

EDEMA OF LARYNX.

Edema of the larynx is characterized by variable edema of the epiglottis and soft palate and by the history of a chronic nephritis or of some local injury or inflammation. The presence of this edema in the course of a nephritis is sufficient to make a diagnosis.

It must not be forgotten that a true edema of the larynx with all of the suffocative symptoms may occur as the result of a severe destructive laryngitis such as occasionally accompanies typhoid fever in which the laryngitis finally involves the cartilages, becoming a true perichondritis.

Aneurism of the Arch of the Aorta.

Aneurism of the arch of the aorta, a mediastinal growth, or a dilated auricle in mitral stenosis, may give rise to a paralysis of the recurrent laryngeal nerve with loss of voice, hoarseness, disturbance of the respiratory rhythm, etc., which are characteristic of laryngitis; hence careful examination of the chest for signs of intrathoracic disease to be followed by an x-ray examination must be undertaken in addition to the necessary laryngeal examinaton of every case of hoarseness, the cause of which is not easily ascertained.

Enlarged Thymus Gland.

In young children an enlarged thymus gland simulates an acute laryngitis. However, there is dullness under the sternum and an x-ray will show a shadow.

C. Diseases of the Bronchi

1. Bronchitis

This condition, inflammation of the bronchial tubes, is acute and chronic in character; it may affect both the large and small bronchial tubes. As a direct result of a chronic bronchitis, a bronchiectasis or dilatation of the bronchial tubes may occur, or there may be asthmatic attacks. The bronchitis may take on a fibrinous character.

Conditions to be Differentiated

These varieties of bronchitis must be separated from each other and from various other conditions, particularly:

Tracheitis
Tuberculosis
Pneumonia
Bronchiectasis
Cardiovascular disease
Emphysema.

Acute Bronchitis.

Acute bronchitis is characterized by cough beginning acutely, and frequently resulting from or accompanying attacks of infectious colds or influenza, or it may be a part of the symptomatology of infectious conditions, such as measles and typhoid fever. There is little or no fever unless the bronchitis is part of a general infection. The physical signs give no *dullness* on percussion.

Over both sides of the chest there are râles usually high-pitched and sibilant in character, heard on both inspiration and expiration. If, however, the finer bronchial tubes are affected the râles are of different character, being fine and crackling and distinct over both sides of the chest. There is often mucous expectoration; there is no change in the percussion note or voice sounds.

The disease, as already stated, is usually a part of the symptomatology of an acute infection, and ends with the cessation of the infection.

Tracheitis.

Tracheitis, which usually accompanies both the bronchitis and the infection, may be distinguished by the fact that while the cough may be of the same type the physical signs over the lungs proper are wanting; râles may be heard or rough breathing may be distinguished over the trachea itself. If laryngitis accompany the condition there is hoarseness, and rough breathing is distinguished over the larynx.

Tuberculosis.

Tuberculosis of the lungs may be mistaken for a simple bronchitis and indeed for a time may be masked by it, but in tuberculosis there is without exception in some part of the lung—usually in the apex—physical signs such as dullness on percussion or change in the breath sounds from mere prolongation of expiration and blowing breathing. Tactile and vocal resonance are increased over the affected area. *Fever*, usually higher at night, is present but very often very slight in degree. Continued loss of weight and cough appear; often there is expectoration in which the tubercle bacilli may be demonstrated, though distinct tuberculosis may be present without the appearance of tubercle bacilli in the sputum. *Anemia* is usually present connected with inability on the part of the patient to do sustained work or exercise.

Continued cough, evening rise of temperature, loss of weight and strength and anemia should arouse suspicion of a tuberculosis of the lungs, even though no physical signs are present.

When bronchitis becomes chronic, especially when it is accompanied by profuse expectoration, the greatest care must be exercised that a tuberculosis is not overlooked. In these cases when there is doubt in the physician's mind, always repeated examination for tubercle bacilli must be made, together with regular temperature measurements and examination with the x-ray. Here as in the acute variety, continued cough, evening rise of temperature, progressive loss of weight and strength and anemia, speak strongly in favor of tuberculosis.

PNEUMONIA.

Pneumonia can be mistaken for acute bronchitis perhaps only by the very careless—but such mistakes have often occurred. There is, however, always fever; there is leukocytosis, even though the physical signs of consolidation which are characteristic of pneumonia do not occur. The patient seems so distressingly ill that suspicion should be aroused. Leukocytosis does not occur in uncomplicated bronchitis.

BRONCHIECTASIS.

Bronchiectasis, which is perhaps invariably accompanied by bronchitis, can be distinguished by the very large amounts of sputum, by the putrid character of the sputum, by the absence of fever, and by the presence of signs of cavity in close proximity to the bronchi. These cases very frequently are accompanied by clubbing of the fingers and they are of extreme chronicity. Here repeated and painstaking observation is necessary to distinguish the cases from chronic tuberculosis of the lungs. Bronchiectasis is chronic and is often accompanied by loss of weight, but there is no fever in the vast majority of cases. Of necessity the presence of tubercle bacilli will make a diagnosis of tuberculosis whether there is bronchiectasis or not.

In this connection must be remembered the occasional difficulty of differentiating certain forms of streptothrix from tubercle bacilli, as pointed out by Gay and Claypole. In children—especially young children under four or five years of age—the von Pirquet tuberculin reaction is of great value.

CARDIOVASCULAR DISEASE.

Cardiovascular disease, giving rise to bronchitis, can be differentiated easily by the presence of disease of the heart and arteries, which are not factors in simple bronchitis.

Pressure on the trachea and bronchi, from a mediastinal tumor, either an aneurism or a new growth, will be diagnosed by dullness under the sternum or along the spinal column, and by the curious unproductive cough and harsh prolonged inspiration and expiration due to pressure.

EMPHYSEMA.

Emphysema can be diagnosticated by the low-pitched prolonged expiration, and the shape of the chest, which is usually of a long anteroposterior diameter with little expiratory excursion.

2. Bronchiectasis

Definition.—Bronchiectasis is a condition in which the bronchi are permanently dilated. The dilatation may be in the form of a sacculation of one bronchus or there may be fusiform dilatation affecting many bronchi.

Symptoms.—The symptoms are cough, paroxysmal in character, with copious expectoration of sputum which may be fetid in character and which is always purulent.

The paroxysms of cough do not occur until a large quantity of sputum collects and the very large amount overflows, or at least comes in contact with healthy bronchial mucous membrane so that cough is excited. Hence the cough often occurs in the morning when arising, when the level is changed and it occurs because the normal mucous membrane is irritated. The sputum is large in amount, often as much as a cupful within a few minutes being expectorated, and often larger amounts within the twenty-four hours; the sputum is offensive, giving a fetid odor to the breath. It often separates into three layers when allowed to stand. The lower layer is thick and purulent, the next above thinner and gray, and an upper still thinner layer frothy and brownish. The sputum may be bloody late in the disease. Clubbing of the fingers is a very frequent accompaniment. There is rarely fever.

The physical signs differ in different cases. In certain cases there are no signs which are indicative of the condition itself; in others there are marked physical signs. If there is a large saccular dilatation there may be dullness over this area, with loss of fremitus and breath sounds. When it is emptied by coughing there is a tympanitic note with bronchophony or pectoriloquy, these signs changing as the cavity fills again. Streptothrix resembling tubercular bacilli may sometimes be demonstrated.

Conditions to be Differentiated from Bronchiectasis

It must be differentiated from:

Tuberculosis
Chronic bronchitis without dilatation of the bronchi
Abscess and gangrene of the lung
Circumscribed empyema.

TUBERCULOSIS.

Tuberculosis is characterized by fever, anemia, sweats, and often by hemoptysis. The large cavities contain sputum full of tubercle bacilli. The tuberculous cavity is surrounded by a dull area due to infiltration of the lung and a bronchiectatic cavity has normal lung tissues surrounding it.

Chronic Bronchitis.

In chronic bronchitis no cavities can be demonstrated. The sputum is less purulent, not so large in amount and does not separate in layers. The cough is more constant.

Abscess and Gangrene of the Lung.

Abscess and gangrene of the lung is more acute; there is high septic fever. The abscess can be localized by the x-ray. The abscess cavity does not fill so rapidly; the case is not remittent. The breath is of a fetid odor.

Circumscribed Empyema.

The collection of pus is generally in the line of the interlobar divisions. It is acute; there is fever and leukocytosis. There is little expectoration unless the empyema ruptures into a bronchus. The rupture like that of abscess occurs suddenly after symptoms which are uncertain.

3. Bronchial Asthma

Symptoms and Physical Signs.—This is characterized by sudden and severe attacks of expiratory dyspnea in which the patient as a rule is more or less suddenly seized with a paroxysm. The sufferer cannot lie down with comfort. The accessory respiratory muscles are brought into play. The patient has a wheezing respiration often heard over the entire house.

Frequently the asthmatic attack is continued over days and sometimes weeks, with more or less severity. The sputum is clear and starch-like and contains Curschman's spirals and eosinophils; the blood shows eosinophils.

On physical examination the patient is seen to be urgently dyspneic, with the dyspnea largely expiratory in type, though there is also difficult inspiration. The chest is acutely distended by the temporary emphysema which exists. The chest is fixed and full of squeaking râles, the expiration being longer than the inspiration. Often the attack is precipitated by proximity to certain animals, by indulgence in certain foods, or by an attack of bronchitis. Some attacks are certainly anaphylactic in origin.

Conditions to be Differentiated from Bronchial Asthma

Asthma may occur as the result of cardiac decompensation or of Bright's disease, and resembles bronchitic asthma in many particulars. It might be mistaken for the asthmatic attack which accompanies and often is a part of vesicular emphysema. It may be mistaken for laryngeal obstruction or for acute bronchitis.

Cardiac Decompensation.

Cardiac decompensation can be distinguished by the fact that in this condition the heart is found diseased; it is usually irregular and dilated. The first sound lacks muscular quality; murmurs may or may not be present. The heart in true bronchial asthma is not affected except secondarily.

There may be expectoration of bloody froth from acute congestion of the lungs. There is always the presence of moist subcrepitant râles perhaps mingled with sibilant râles, but the moist râles are not present in bronchial asthma. In paroxysmal cardiac asthma the chest is full of moist râles, expiration is not prolonged and there is not the expiratory disturbance which is so characteristic of bronchial asthma.

Bright's Disease.

In the asthma of Bright's disease the physical signs of bronchial asthma obtain, but there is the presence of albumin or casts in the urine; the heart is hypertrophied and there is high blood pressure. In these cases, too, there is apt to be cardiac decompensation.

Simple Emphysema.

From simple emphysema, the attack is distinguished by its peculiar characteristics of sudden severe dyspnea. In the absence of attacks of bronchitis, the emphysematous individual is constantly dyspneic and there are the physical signs of distention of the chest, diminution of the heart dullness, and very prolonged expiration. Bronchial asthma is characterized by the attacks being paroxysmal in type.

Compression of the Bronchi or Trachea.

Compression of the bronchi or trachea by enlarged bronchial glands, by enlarged thymus, aneurism and mediastinal growths gives rise to a characteristic wheeze. The expiration is not alone prolonged. Auscultation over the trachea will show a high-pitched inspiration and expiration of about equal length. Physical examination of the chest will prove the presence of dullness over the bronchi or trachea and in addition the other signs of aneurism when compression is due to that condition. Examination by the x-ray will show a shadow under the sternum.

Hysteria.

Hysteria is sometimes accompanied by a dyspnea, but there are no râles, nor is there prolonged expiration. The patient has the typical condition of a hysterical individual; the breathing is sharp and jerky.

ACUTE BRONCHITIS.

In acute bronchitis there is often prolongation of expiration, but the dyspnea is not urgent; there are apt to be signs of inflammation of the respiratory tract further up, such as rhinitis and trachitis.

FOREIGN BODIES.

Foreign bodies in the larynx, trachea, or bronchus, or a new growth in the trachea or larynx, give rise to constant unproductive coughing; a laryngoscopic or bronchoscopic examination will indicate the local condition.

4. Fibrinous Bronchitis

(*Plastic Bronchitis—Croupous Bronchitis*)

This condition is chronic or acute, the former being more common.

Symptoms.—It is characterized by dyspnea, cough and the expectoration of casts of the bronchi.

Occurrence.—It occasionally occurs during diphtheria, being more common with this condition before the use of antitoxin. The author has notes of a case where the casts were present to the third degree of fineness. The expectoration of the casts is diagnostic.

In the chronic cases, the paroxysm of dyspnea, cough and expectoration of casts occur at intervals, separated by weeks or months. There may be fever and hemorrhage with the attacks.

Conditions to be Differentiated from Fibrinous Bronchitis

It can be confused with:

HEMOPTYSIS, in which there are blood casts of the bronchi, but these are blood clots and not fibrinous material.

ASPERGILLOSIS.—Here the fungi are expectorated in castlike masses, but the microscope will show the fungi.

In certain cases of pneumonia and acute bronchitis, there are casts of the bronchi, which are distinctive of the conditions.

D. Diseases of the Lungs

1. Congestion of the Lungs

Etiology.—Congestion of the lungs may be active or passive. It is the result of one of direct obstruction to the vessels of the lungs, or it may be due to weakness of the heart, the latter either the result of myocardial change due to weakness during an acute infection, to valvular disease, or to a primary cardiac sclerosis.

Symptoms.—The symptoms are cough, dyspnea, more or less expectoration, with physical signs of dullness somewhat increased, tactile and vocal fremitus and subcrepitant râles.

Conditions to be Differentiated from Congestion of the Lungs

This condition may be mistaken for:

Pneumonia
Pleural effusion
Temporary atelectasis.

PNEUMONIA.

Congestion of the lungs may be distinguished from pneumonia by absence of fever, pain, leukocytosis and by the absence of pneumococci from the sputum and blood culture. To these must be added the important fact of the presence of cardiac lesion and the fact that the patient has been ill and confined to bed, thus allowing a collection of fluid to collect in the air vesicles of the lungs.

PLEURAL EFFUSION.

In pleural effusion there is the same lack of acuteness of the symptoms, but in pleurisy there is absence of tactile fremitus and resonance and absence of voice sounds. The dullness is movable by change of the position, and there is dislocation of the heart and other adjacent organs. Puncture with a needle will prove the presence of liquid.

TEMPORARY ATELECTASIS.

Temporary atelectasis might be mistaken for passive congestion because there is dullness over the portion of the lung involved, but a few long breaths, if the individual is an adult in good health, will decide the question beyond danger of mistake. If the patient is an infant, the shock, the local sign of dullness, etc., will help to easily make a differential diagnosis.

2. Edema of the Lungs

Chronic congestion of the lungs, edema due to cardiac decompensation, has been described.

Acute edema of the lungs occurs as the sudden result of cardiac decompensation, or of high blood pressure as the result of an acute infection, and in cases of paroxysmal tachycardia and mitral stenosis. It is often the result of the latter condition and follows some unusual exertion such as running, lifting or walking in a high wind, and during labor. Stengel has described a paroxysmal variety in which the patient, always with some arterial, cardiac or renal fault, is seized without warning, often after resting, with a series of characteristic symptoms—urgent dyspnea, sometimes the heart being full and regular, sometimes rapid, sometimes with the chest full of moist râles, expectoration of huge amounts of bloody froth, the case ending almost instantly upon administration of a hypodermic of morphin.

The writer has seen several such cases. One old lady regularly had three or four such attacks a year, the attacks always occurring during the night, and until the fatal attack they were always easily relieved.

The condition in the first class of cases is often the final scene of acute cardiac dilatation, in others of paroxysmal tachycardia; the last condition is constantly mistaken for acute dilatation. There is probably always some cardiac dilatation, but it can be differentiated from edema due to this cause by the area of cardiac dullness being relatively little increased, and by the much more prompt relief by the administration of morphin.

3. Pneumokoniosis

Cause.—This condition is the result of inspiring much dust.

Occurrence.—It is common in stone cutters, miners, iron grinders and more frequent in dwellers in cities than those who live habitually in free country air and in the woods.

It is constantly connected with chronic bronchitis, emphysema and tuberculosis in miners and stone workers.

Symptoms.—In certain cases years after the individual has ceased to work in the mines he expectorates large amounts of dark—almost black—purulent sputum. Such individuals as a rule have upon their faces and hands pigmented scars, which also mark their occupation. The mere deposit of the pigments and dirt in the lungs gives rise to no symptoms save dark sputum for years, indeed, none at all unless the deposit of dust has caused some irritative condition.

Diagnosis.—The character of the sputum makes the diagnosis. There is nothing distinctive about the lung lesion itself, but where emphysema, bronchitis and tuberculosis are present with black sputum, the diagnosis should certainly be pneumokoniosis.

4. Hydrothorax

Cause.—Hydrothorax is the collection of serum in the pleural cavity, the result of cardiac decompensation or pressure on the vessels by a tumor, or it may be due to aneurism.

Physical Signs.—There are the physical signs of a freely moving liquid in the chest accompanying any one of the above conditions (See serofibrinous pleurisy). It is apt to be bilateral, though in aneurism it is most frequent on the right side.

There are no symptoms except dyspnea, dislocation of the organs, and possibly increased cardiac disturbance, which are directly referable to the liquid.

Conditions to be Differentiated from Hydrothorax

It must be differentiated from:

Pleurisy with effusion
Edema of the lungs.

Pleurisy With Effusion.

In serofibrinous pleurisy there are symptoms of inflammation which are wanting in hydrothorax; pleurisy is apt to be unilateral.

Edema of the Lungs.

In edema of the lungs (hyperstatic congestion) there is stationary dullness (not so marked as over a liquid); there are more evident breath sounds and voice sounds; tactile fremitus is better preserved, and there are numerous moist râles, which are never present over a liquid.

5. Hemoptysis

Causes.—Spitting of blood occurs from many different causes, all of which must be considered before a diagnosis can be certainly made. Its differentiation from hematemesis has been discussed on page 281.

The most common cause of spitting of blood is *tuberculosis of the lungs.* In every case this condition must be fully considered before it is dismissed. The proper state of mind for the physician in regard to spitting of blood is to *think first of tuberculosis,* and then proceed to make a differentiation, by examining one organ after another—the lungs included—which may be responsible for hemoptysis. Nothing is more harmful than lack of examination in hemoptysis, and the attributing of many cases to bleeding from the throat. This is the unfortunate habit of conversation, if not of thought, of many physicians. While dilated pharyngeal veins may cause spitting of blood, it is not common.

Hemoptysis may be due to:

1. Epistaxsis.—This can be differentiated by examining the nose and pharynx. A bleeding point can be seen in the nose, and often the

blood can be seen trickling into the pharynx, or clots in the pharynx if blood comes from the nose. Care must be taken to eliminate clots which have lodged in the pharynx or nose from blood coughed up.

2. Bleeding from the pharynx occasionally occurs, and reports of a ruptured vein in the throat giving rise to profuse bleeding have been made. This must be very rare. Bleeding from the pharynx much more frequently is in small amounts, and comes from erosions or ulcers, which can be seen. If the point of bleeding cannot be ocularly demonstrated, the diagnosis of pharyngeal hemorrhage should not be made.
3. Bleeding gums or tongue. Often in diseased conditions the gums bleed as a result of the least disturbance; the tongue may be bitten, or may be ulcerated. Both of these conditions can be seen.
4. Bleeding from the larynx. This occurs as the result of ulceration or traumatism. It is often accompanied by hoarseness. The diseased bleeding area can be seen by means of the laryngoscope.
5. Bleeding from the trachea and bronchi may occur as the result of ulceration of these parts. One must be sure of the non-existence of tuberculosis before such a diagnosis is made. The ulcer may be seen by the expert use of a bronchoscope.
6. Bleeding from the lungs in pneumonia. The blood is rarely profuse; the sputum is rusty. If profuse bleeding occurs with what appears to be a lobar pneumonia, it is extremely probable the case is tuberculous in origin.
7. Bleeding from emphysema and bronchiectasis, in the absence of tuberculosis, occasionally occurs. Here, too, however, great care must be taken, by repeated negative examinations of the sputum, x-ray examinations, and so forth, before one excludes tuberculosis.
8. Aneurism of the aorta may rupture into a bronchus, causing either bloody sputum at first, followed by profuse bleeding and death, or rapid profuse bleeding in the beginning. Signs of aneurismal dilatation of the aorta must be carefully searched for by examination with the x-ray, which is one of the most valuable methods of differentiation.
9. Bleeding in septic states, in purpura, in hemoptysis and certain cases of jaundice and poisoning occur. Here the causative factor is usually patent and should be recognized.
10. Vicarious hemorrhages at the time of suppressed menstruation have occurred, and cases apparently well-authenticated by the absence of lung lesion are on record. In the author's mind, however, most—indeed practically all—cases of supposed vicarious hemorrhage in young women with suppression of menstruation are really cases of

tuberculosis with suppression of menstruation as one of the symptoms, the lung lesions being extremely meager. It would perhaps be well to banish the term, or at least to look upon vicarious hemorrhage from the lungs as one of the rarest happenings in medicine.

11. Osler in his practice, under this heading speaks of sudden hemoptysis occurring in young persons, continuing for a few days and disappearing, leaving no traces. These facts are unquestionably correct, but what assurance is there that the cases were not due to a small tubercular focus giving no physical signs, and entirely clearing up without any continuation of ill health to the individual? It would be better for the future safety of such patients to consider them tuberculous.
12. Heart lesions, especially mitral stenosis, are prolific sources of hemorrhage from the lungs; careful examination will always reveal the cardiac defect. In certain cases where there is an old pulmonary lesion and a mitral stenosis in which the heart is losing compensation it is difficult to determine which is responsible for the bleeding, the lung lesion or the heart lesion; usually the case may be diagnosed by careful attention to the symptoms. If the signs of decompensation are in the ascendant, then it is probably the heart lesion which is at fault; if on the other hand there is rapidly increasing lung disease, usually the lung can be looked upon as the cause of the bleeding.
13. Abscess of the lung, gangrene of the lung, malignant disease, mycosis, distomatosis and rupture from a subdiaphragmatic abscess will often cause a hemorrhage. The cause can be diagnosed by the symptoms and physical signs.
14. Hemoptysis, following heavy lifting, is more often than not bleeding from a tuberculous focus which has been disturbed.
15. *Last and most important:* hemorrhage occurs from the lungs as an early or late symptom of tuberculosis of the lungs. The blood is coughed up, it is usually frothy, though in cases of severe bleeding the blood may be so profuse as to appear clotted and comes from both mouth and nose. A common symptom is a sweet taste in the mouth followed by cough and the appearance of frothy blood. When the hemorrhage is severe, the initial attack is frequently followed by coughing up bloody sputum for several days. Usually an area of consolidation can be observed in the lung. It is often difficult, however, to certainly note the spot in the lung from which the bleeding comes. When no area of consolidation can certainly be made out, there can usually be found symptoms of anemia, slight fever, loss of weight or inability to do the usual work, which will point to tuberculosis.

6. Chronic Pneumonia

Chronic pneumonia may be of the interstitial type, of the bronchial type, or it may originate from a chronic pleurisy.

Symptoms and Course.—With this condition the general health is good; it is chronic in its course. There is cough, usually with much expectoration; there is little emaciation. Hemorrhage is said to occur. The physical signs are those of retraction of the chest with signs of consolidation.

Conditions to be Differentiated from Chronic Pneumonia

The diseases with which it may be confounded are:

Tuberculosis
Bronchiectasis
Syphilis of the lung
New growth of the lung.

TUBERCULOSIS.

The physical signs may be in no way different from those of tuberculosis. The differentiation must therefore depend upon several factors. Tuberculosis in active state always has an evening rise of temperature; if the tuberculous process, however, is well walled off, fever will not be present. Marked and continued emaciation favors tuberculosis, as does also much expectoration with or without cavity formation.

Hemoptysis will speak almost surely for tuberculosis. There is no other condition of the lung itself which so constantly gives rise to hemoptysis.

The presence of signs of cavity not in close proximity to a bronchus will favor tuberculosis.

The presence of tubercle bacilli is a positive sign of tuberculosis.

BRONCHIECTASIS.

Bronchiectasis may be differentiated by the great amount of sputum, often of fetid character, the appearance of signs of cavity with the expectoration of large amounts of sputum, and again the disappearance of the cavity when it has been present a short time before.

The absence of emaciation, the long duration of the case, and the absence of tubercle bacilli will help to differentiate this cavity from the tuberculous one. In the presence of large amounts of expectoration, tubercle bacilli may be difficult to demonstrate. Care should therefore be taken to dissolve the sputum with some such reagent as antiformin.

For the details of this process, the reader is referred to works on clinical miscroscopy.

SYPHILIS OF THE LUNG.

Syphilis of the lung may give the same physical signs as chronic pneumonia, but the former gives the Wassermann reaction and there is usually a history of secondary and primary lesions.

NEW GROWTH OF THE LUNG.

Tumors of the lung may simulate in physical signs a chronic pneumonia, but the case is of much shorter duration, the areas of infiltration are apt to be disseminated, and a primary growth may be discoverable. The seat of the primary growth may of course be almost anywhere in the body, but the uterus and the rectum must never be forgotten as possible infecting areas.

7. Emphysema

Definition.—Emphysema is a dilatation of the air vesicles with or without atrophy of the alveolar walls.

Acute and Chronic Emphysema.—It may be acute or chronic. The *acute variety* is the result of a sudden bronchial spasm such as is present in acute asthma or it may be present in the lungs of those dying of capillary bronchitis. In milder degrees, compensatory emphysema is present in such conditions as massive pneumonia of one lung when the lung of the opposite side undergoes a compensatory enlargement. This same condition occurs when a pleural effusion or pneumothorax suddenly or gradually disables one lung in the mechanism of respiration. This acute or compensatory emphysema is recognized chiefly by the physical signs.

There is enlargement of the side of the chest not affected by the disabling lesion. There is hyperresonance and prolongation of expiration, not blowing in character, over the emphysematous lung. The breath sounds are apt to be louder than normal; there is no dislocation of the viscera.

Conditions to be Differentiated from Unilateral Emphysema

The one possible condition with which unilateral emphysema might be confused is:

PNEUMOTHORAX.

Pneumothorax is unilateral. It dislocates the other viscera to the opposite side and downward. The note over a pneumothorax is tympanitic; in rare instances the note is dull; there is absence of breath sounds over the collapsed lung and of tactile fremitus and voice and breath sounds. If there is a pyopneumothorax there will be the coin sound and the signs of effusion at the base of the chest with a succussion splash.

Hypertrophic Emphysema

This is the form of emphysema of the lungs in which the whole organ is larger than normal. The vesicles are distended, the alveolar walls are thinned and atrophied.

Cause.—The cause of the condition is overuse of the lungs, such as occurs in players of wind musical instruments, in glass blowers, and diseases such as chronic bronchitis and prolonged or recurrent attacks of asthma.

Symptoms and Physical Signs.—The symptoms are continued dyspnea on exertion, cough, and cyanosis.

The physical signs are distended thorax with increased anteroposterior diameter, hyperresonance, decreased tactile fremitus and prolonged expiration. During an acute attack of bronchitis or asthma, the prolonged expiration is very easily distinguished because of the accompanying râles. When there is no asthma or the bronchitis is quiescent, the prolonged expiration is difficult to distinguish on account of its extremely low pitch. Therefore, one must keep this fact in mind and watch must be kept for this condition. The heart dullness is often obscured by the emphysematous lung tissue. In later stages cardiac decompensation occurs with all the symptoms of cardiac decompensation due to any other condition.

Conditions to be Differentiated from Emphysema of the Lungs

The three diseases with which the condition is confounded are:

Tuberculosis of the lungs
Cardiac decompensation
Tumors.

TUBERCULOSIS OF THE LUNGS.

The differentiation between tuberculosis of the lungs and emphysema should scarcely require mention, except in those cases of emphysema which are accompanied by bronchiectasis. Here the dyspnea and the expectoration of large amounts of purulent material lend some color to the diagnosis of tuberculosis. However, the presence of tubercle bacilli in the sputum, the sweats, the fever and the emaciation, the presence of dullness often at the apex with blowing breathing, make the diagnosis absolute for tuberculosis.

CARDIAC DECOMPENSATION.

Cardiac decompensation resembles emphysema only in its dyspnea, but in the later stages of emphysema, cardiac decompensation occurs as the direct result of the diseased lung, causing dilatation of the right heart. When this occurs there is emphysema plus cardiac decompensation.

The differentiation can only be made by the history and the physical signs of the case. The history of a long-standing cough accompanied by asthma without any actual cardiac disease, absence of harsh murmurs over the heart, together with dilatation of the cavities of the heart, will point toward emphysema as the cause. On the other hand, the history of cardiac palpitation, of edema which has been of long standing, together with harsh murmurs over the body of the heart which have been present before the cough, will serve to decide the case as being cardiac in origin.

TUMORS.

Tumors pressing upon the trachea or upon the bronchi sometimes cause breathing which resembles somewhat that of emphysema, but the lung whose bronchus is compressed is totally or partially collapsed, the expiration is high-pitched, the inspiration is more affected than the expiration and dullness over the tumor can be discerned. There is not the same history of prolonged dyspnea with recurrent attacks of bronchitis and asthma as there is in emphysema.

8. Gangrene of the Lung—Abscess of the Lung

These two conditions will be considered jointly, because their course and symptoms are very similar. While the prognosis of the former is more serious than that of the latter the chief points of differentiation between them have to do with the *character and degree of destruction of the lung tissue.* Both of these conditions are the result of destructive processes in the lung, the result of a pneumonia of a septic infarct or an infarct which has become septic.

Symptoms.—They are characterized by irregular fever, sweats, depression, leukocytosis and signs of local consolidation in one or another part of the lung. An x-ray examination will show a shadow at the site of the collections.

When the abscess opens into the bronchus there is much expectoration of purulent material. If there is gangrene this material is of the peculiar sickening stench which is characteristic of gangrene.

Conditions to be Differentiated from Gangrene and Abscess of the Lung

The condition must be differentiated from:

Empyema, both free in the pleural cavity, and localized between the lobes of the lung
Tuberculosis
Bronchiectasis
Abscess of the liver
Suppuration of peribronchial glands.

EMPYEMA.

Empyema has the same subjective symptoms as abscess or gangrene, fever, sweats, leukocytosis and progressive loss of strength and weight. The physical signs of free and localized empyema differ. In free empyema there is dullness over the whole affected side, which may be movable on changing the position of the patient. The viscera are dislocated, the heart being pushed to the opposite side; the liver depressed if the collection is over the right side; the stomach tympany in the lower left chest is diminished or absent if the collection is on the left side. There is loss of tactile fremitus and vocal resonance over the collection of fluid. If the empyema is sacculated, and this is the form which is most difficult to differentiate from abscess or gangrene, there is dullness which does not vary on change of position of the patient. There are all the signs of a collection of fluid, and not consolidation of the lung. The x-ray helps to localize a collection of liquid; if this collection is in the line of one of the divisions between the lobes the likelihood is that it is due to a localized empyema, but if the collection is or seems to be in the substance of the lung, the point is in favor of abscess. Then, too, the history is a help; if there is a clear history of pneumonia the chances are the condition is a collection between the lobes or one localized between the lower lobe of the lung and the diaphragm. If the case is seen after an empyema has ruptured into a bronchus, the diagnosis must depend largely upon the finding of dullness over the old area, and especially upon its persistence at the base of the lung. An abscess is likely to empty its cavity at once, and if recovery takes place leaves practically no physical signs behind.

If the lung condition, however, follows a septic disease such as septicemia, it is probable that the condition is an abscess. The insertion of a needle will help to make a diagnosis of a fluid collection, but will not help to distinguish between an abscess and an empyema.

The chief point, therefore, is the *area affected*, and as to whether it is in the lung tissue or whether it is simply localized between the lobes of the lung.

TUBERCULOSIS OF THE LUNG.

Tuberculosis of the lung is also confined to a particular portion of the lung, but here, though the process is localized, the chances frequently are that the localization is in the apex. There is usually *increased* fremitus and vocal resonance, instead of diminished. If pus is expectorated there is, as a rule, no appearance of a sign of a cavity. The disease is progressive. There is usually a history of failure of health before the local signs appeared and there is the presence of tubercle bacilli in the sputum.

Much difficulty may be encountered if the condition has gone on to a rapid cavity formation and the walls of the cavity have become gangrenous, but here the history will be of great value. Almost always in tuberculosis a history of ill health can be obtained which was present before acute symptoms have occurred.

BRONCHIECTASIS.

A large bronchiectatic cavity filled with pus might in the first examination be mistaken for an abscess, but the condition is one of *chronic* character. The cavity is cleared soon after a spell of coughing, and refills rapidly. There is not nearly so much general depression, while the expectoration is foul-odored; there is not the marked gangrenous character to the odor.

ABSCESS OF THE LIVER.

Abscess of the liver may rupture through the diaphragm and give rise to expectoration of pus. In this condition there is usually an antecedent history of dysentery, a long history of hectic fever not connected with any lung condition; there is enlarged liver. Usually there is a history of residence in a tropical or subtropical region. The expectorated material is likely to be prune-colored, due to changed blood, and Entamebæ histolytica can be demonstrated. All these symptoms differ from those of an ordinary abscess of the lung.

SUPPURATION OF PERIBRONCHIAL GLANDS.

Suppuration of the peribronchial glands may simulate an abscess of the lung, but there is wanting in the history the usual reasons for a lung abscess, to wit: injury, pneumonia or septic infarct. Then the collection as seen by the Röntgen ray is found near the root of the lung, which is unusual in the ordinary abscess of the lung.

9. New Growths of the Lung

Carcinoma, sarcoma and, occasionally, *adenomata* are found as tumors of the lung. Occasionally they are secondary to a growth somewhere in the body, rarely are they primary. There is no possibility of differentiating between sarcoma and carcinoma unless the nature of a primary growth in some other portion of the body is known.

Symptoms.—The symptoms of this condition are those of encroachment on the circulation, or aëration of the lung, or intrathoracic pressure. There is cough, cyanosis, dyspnea; pain is present if the growth encroaches upon the pleura and presses upon the vessels in the thorax. Sometimes the growth is so large that there is displacement of the organs, of the heart and the liver especially. Frequently there is pleural effusion.

The physical signs consist of dullness over the area of the growth; sometimes the sign of pleural effusion, whether a true effusion is present or not. There may be signs of compression of vessels at the root of the neck and the trachea or esophagus, the result of the growth being in the upper portion of the lung and making pressure there.

The symptoms of the form which affects the parenchyma of the lung, are gradually increasing dyspnea, with cough, and frequently sputum, which is bloody and contains much mucus. There is dullness on percussion over the affected area, but this dullness is often not continuous, depending upon the fact that there is often unaffected lung tissue between the new growths. If the tumor invades the pleura, then there are the signs of pleurisy, and often the condition is later accompanied by effusion into the pleural cavity. If the growth encroaches upon the mediastinum, the symptoms are those of pressure on the trachea and great vessels.

Conditions to be Differentiated from New Growths

The condition must be differentiated from:

Pneumonia
Pleurisy which is non-malignant
Tuberculosis of the lung
Actinomycosis
Mediastinal disease
Aneurism of the arch of the aorta.

PNEUMONIA.

Pneumonia is acute. It has a history of chill and fever, accompanied by a sudden involvement of one or more lobes of the lung—the lower lobe is usually affected first. There is acute delirium, frequently with rapidly rising leukocytosis; in a word, all the signs of an acute infection.

In tumor the onset may occasionally be sudden, but there is never the rapid involvement of the lung which characterizes pneumonia. The physical signs are limited in the early stages to small areas, and could scarcely be mistaken for the consolidation of pneumonia. The sputum of pneumonia is filled with pneumococci, while these do not appear in new growths or are few in number.

PLEURISY.

Frequently when the case is first seen or attention is first drawn to the chest, there is complete dullness over the chest with dislocation of the neighboring viscera and disappearance of breath and voice sounds; in other words, the signs of pleural effusion. In other cases there is a sudden pleuritic pain which is the result of a pleurisy secondary to a new growth. On tapping a pleural effusion due to a new growth it is frequently found to be bloody and to contain many epithelial and round cells; on removal of all of the liquid, signs of the consolidation of the lung underneath are found. Cytodiagnosis is of the greatest importance in making a differential diagnosis. If there is pleurisy without any effusion there is pain, dyspnea, restricted breathing and all the signs of a local inflammation of the pleura. This cannot be differentiated from a simple pleurisy unless examination of the case at that time, or on some previous examination, showed signs above stated of a consolidation and a history of growth in the lung.

TUBERCULOSIS OF THE LUNG.

Tuberculosis of the lung, by its insidious beginning, by the fever, emaciation and anemia, together with the physical signs of consolidation, might easily be mistaken for a new growth. But the history of the case, the sputum containing tubercle bacilli, the location of the lesion most frequently at the apex, the signs of consolidation without increase of size of the lung, the formation of excavation—all help to distinguish the conditions.

ACTINOMYCOSIS.

Actinomycosis may simulate a malignant new growth in all particulars except perhaps one—that it is much more chronic. If there is sputum or if a thoracentesis is done, then the peculiar organism characteristic of actinomycosis will be found.

MEDIASTINAL DISEASE.

Mediastinal disease may be simulated by a growth in the lung when the tumor is at the apex and encroaches upon the upper portion of the mediastinum. Only the fact that previous symptoms or physical signs above described have enabled one to make a diagnosis of a tumor of the lung, of which this set of symptoms is a part—cyanosis of the face, dullness under the manubrium and difficulty of swallowing, will help to differentiate the two conditions.

Aneurism of the Arch of the Aorta.

A tumor of the lung which has encroached upon the vessels and tissues in the upper mediastinum may be confused with aneurism. Especially might this question come up when the vessel involved is the lower end of the jugular or the subclavian artery in any of its portion. In aneurism there is dullness on percussion, but there will be pulsation, possibly a murmur or thrill over the dilatation; a diastolic shock will also be felt. None of these are present in a tumor. The pulsation of aneurism is expansile in character while that of tumor is simply a transmitted pulsation. However, the differentiation needs all the possible care that can be given to such a case.

In aneurism there is no enlargement of the lymphatic glands. A fluoroscope will show the expansile pulsation of an aneurism; the shadow of a new growth can be seen, it does not pulsate.

10. Pleurisy

Various Forms.—Acute, chronic and purulent pleurisy are the forms in which this condition is found.

(*a*) *Acute Pleurisy*

Acute pleurisy is the result of infection of the pleura due to various organisms, pneumonia and tuberculosis being the most common. Tubercular pleurisy has been considered under the subject of tuberculosis.

Characteristic Features.—Acute pleurisy is characterized by severe sharp pain over the chest on the side affected or in the abdomen, if the diaphragmatic surface or lower portion is particularly involved. There is fever usually of slight degree, a friction sound, very slight impairment of resonance, together with slightly lessened vocal fremitus. Movement is decidedly limited on the affected side.

Conditions to be Differentiated from Acute Pleurisy

The condition may be mistaken for:

Pneumonia
Pleurodynia
Intercostal neuralgia
Traumatism
Appendicitis.

Pneumonia.

Pneumonia is constantly accompanied by a pleurisy. The usual error is to consider a beginning pneumonia as merely a pleurisy. In the very earliest hours the physical signs of a beginning pneumonia may be limited to those of pleurisy, but the general condition is much more severe. The patient has more fever; there is more leukocytosis; there is apt to be rusty sputum, and very soon the signs of consolidation occur with dullness and blowing breathing.

PLEURODYNIA.

Pleurodynia, a simple myositis of the chest, often gives severe pain upon deep inspiration, but there is tenderness upon movement and upon pressure. In pleurisy pain upon movement is present following a peculiar kind of motion. There is soreness on pressure over a pleurodynia. The physical signs of pleurisy are entirely wanting.

INTERCOSTAL NEURALGIA.

Intercostal neuralgia or neuritis is due either to an infection or to pressure from a growth, or to disease of the spinal column.

There is great pain upon pressure over the affected nerve or nerves, the most painful spot being at the posterior angle of the ribs, at the midaxillary line and at the anterior extremity of the ribs which are the points of exit of the skin branches of the nerves.

There is no fever, nor is there any other constitutional symptom unless the neuritis is the result of an infection or some such disturbance as caries of the spine, which causes great fever, loss of weight, etc.

TRAUMATISM.

A traumatism might be mistaken for a pleurisy where no history is present of an accident, but the history will clear up the case. It must be remembered, however, that a traumatism of the chest may give rise to pleurisy, which will then be recognized by the physical signs; an x-ray will often decide the case. The author has notes of a case where no history of a blow could be obtained; there was tenderness. An x-ray showed a broken rib.

APPENDICITIS.

Appendicitis may be simulated by a pleurisy because of the distribution of the pain to the appendiceal region, but in appendicitis there is local resistance and tenderness over the appendix. This is not usually the case in pleurisy. Rectal examination will often show a tender mass in the pelvis which is not present in pleurisy.

Conclusions

The entire lesson to be learned from these conditions is that *one must never consider a simple pain in the side pleurisy without careful physical* *examination,* and on the other hand pleurisy, which can be recognized only by a careful physical examination, must not be overlooked for want of such an examination.

(*b*) *Serofibrinous Pleurisy—Pleurisy with Effusion*

Etiology.—Serofibrinous pleurisy, or pleurisy with effusion, is the result of an exudate following a simple fibrinous pleurisy. It may be latent in its beginning—a chest becoming full of liquid with practically no symptoms of which a history can be obtained, the first complaint of the patient being dyspnea. Other cases directly follow the onset of an acute pleurisy above described. It is frequently tuberculous in character.

The character of the liquid may be well demonstrated by examination of the liquid. Tubercle bacilli may be demonstrated; a great excess of lymphocytes speaks for tuberculosis, but animal experimentation will give the final test. If an animal is inoculated and develops tuberculosis, it is positive proof of the tuberculous character of the effusion. Bloody liquids are likely to be either tuberculous or malignant. Endothelial cells are probably due to a transudate.

Symptoms and Physical Signs.—The symptoms and physical signs of a pleurisy with effusion depend entirely upon the severity of the preceding symptoms and upon the amount of fluid present in the chest—principally upon the latter. There is usually fever of slight degree, depending upon the severity of the initial infection. The other general symptoms of infection—depression, loss of appetite and failing strength—are also present.

The effusion, as observed in the beginning, gives rise to dullness which is movable upon changing the position of the patient; the dullness first appears at the base of the chest affected. This dullness gradually increases. There is loss of tactile fremitus and voice sounds over the area. With increase of the liquid, the area of abnormal sounds increases in extent. Gradually as the fluid increases, and the pleural cavity is encroached upon, the neighboring organs are dislocated—the heart to the right or left, the liver downward, and the stomach tympany diminishes or disappears from the lower left chest. Occasionally there is high-pitched voice and breath sounds—Bachelli's sign—over the liquid. There may be high-pitched voice sounds, egophony, which are quite characteristic of liquid, but there is always diminished or lost fremitus. The chest is usually bulging on the side affected and there is less movement than on the normal side. When there is a pleural effusion on one side, there is usually a triangular area of dullness at the base of the opposite chest, the base of the triangle being at the lower part, the vertebral column forming the altitude, and the hypothenuse being formed by the line joining the two: this is the Grocco's sign.

The lung on the affected side is compressed at the apex. There is often a dull note with blowing breathing and bronchophony over the compressed lung. A radiograph will show a shadow in the position of the liquid and will also show the dislocated heart (Fig. 65).

If the liquid becomes purulent, there is increasing leukocytosis with septic fever and great emaciation of the patient. Over purulent collection there is rarely or never increased voice sounds or blowing breathing in the adult. In children, however, there may be blowing breathing and increased breath sounds over a purulent effusion.

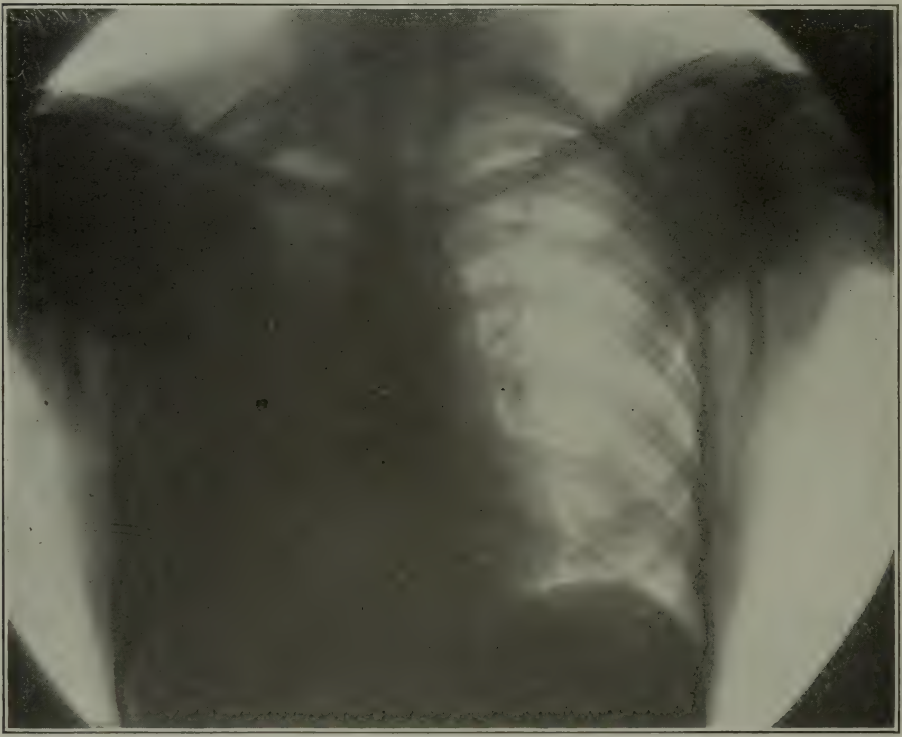


Fig. 65.—X-ray Picture of Pleural Effusion.
H. K. Pancoast fecit. (Original Observation.)

When the liquid is encysted there is marked variation in these signs; there is, however, almost without exception, loss of fremitus and voice sounds, with complete dullness over the area marked by the effusion.

Conditions to be Differentiated from Serofibrinous Pleurisy

Thickened pleura
Consolidation of the lung (pneumonic or tubercular)
Edema of the lung (hypostatic congestion)
New growth

Abscess
Transudate due to heart decompensation
Aneurism
Extremely large liver.

Thickened Pleura.

In certain instances a thick pleural membrane will give rise to dullness, decreased fremitus, and diminished breath and voice sounds, but there is no flatness as there is in effusion; the breath sounds are only slightly altered and the paravertebral dullness is not present.

Consolidation of the Lung.

From pneumonia the condition is separated by the history of the case and by the physical signs. In pneumonia the attack comes on frequently with a chill, high fever, leukocytosis, and usually with rapid involvement of one or more lobes of the lung. There is dullness, increase of fremitus and voice sounds, dullness following the line of the lobe or lobes of the lung. The dullness appears within twenty-four to forty-eight hours, while that of pleural effusion comes on much more slowly.

In those cases which are not extremely rare, where the voice sounds and breath sounds are exaggerated over a pleural effusion, there is no fremitus and there is dislocation of the viscera. An exploratory puncture will settle the question as to whether the physical signs are due to a liquid or to consolidation. Care must be taken, however, to see that the needle is inserted far enough into the chest. Frequently there is a small amount of liquid between the chest wall and the lung which may be withdrawn, showing the presence of liquid, but when aspiration is undertaken no large collection will be found.

Edema of the Lung.

In hypostatic congestion or passive edema of the lungs the symptoms of chill, fever and leukocytosis are wanting, and one may be at a loss to properly interpret the physical signs. However, in the edema there is slight fremitus present, there are usually subcrepitant râles and there is no displacement of the organs as there is in a collection of liquid. The exploratory needle attached to the syringe will establish the diagnosis. No fluid is found in edema.

Perhaps the greatest difficulty arises in differentiating an encysted collection of fluid from a consolidation. After pneumonia there is frequently left a collection of fluid encysted between the lobes of the lung or toward the mediastinum, or below the base of the lung. When in the latter position it is difficult to tell whether the collection is above or below the diaphragm. The difficulty of diagnosis here arises between an abscess or gangrene of the lung and a collection of pus between the lobes. Often after the most careful needling of the lung, a liquid cannot be demonstrated.

Under these circumstances a diagnosis is best made by locating the liquid with the aid of an x-ray and then aspirating at the known position. The shadow of a collection of pus, too, is much more dense than that due to a consolidated lung. The diagnosis between an interlobar collection and an abscess must be largely made by the position of the collection or shadow. When the collection is between the diaphragm and the liver, difficulty will be had as to whether the collection is above or below the diaphragm. Here, however, the x-ray is of the greatest value. If the collection is below the diaphragm it will usually move with the diaphragm independently of the lung. The history of appendicitis or suppurative renal condition, the greater prominence of abdominal symptoms in subdiaphragmatic collection is of value as indicating a subdiaphragmatic collection, while the chest collection is almost without exception preceded by a history of pneumonia or pleurisy.

New Growth.

A new growth has some signs of a collection of liquid but differs in point of history. A growth is usually slow in development, a liquid is more rapid. If the liquid is due to a tubercular process the difficulty may be greater because of the more chronic character of the tubercular as compared with the septic process.

Transudate Due to Heart Decompensation or Aneurism.

A transudate due to cardiac decompensation or pressure from enlarged glands or a new growth of an aneurism can be mistaken for an effusion due to pleurisy. Here the physical signs of the effusion are identical, but in pleurisy there is always a history of infection even though the symptoms have been mild. In the transudate there is the history and physical signs of cardiac derangement. Usually there is a preponderance of epithelioid cells in the exudate due to pleurisy—either lymphocytes or polymorphonuclear cells are present in large numbers.

Occasionally a sacculated purulent effusion pulsates synchronously with the heart, resembling thereby an aneurism. In aneurism, however, there is tracheal tug, diastolic shock, often a murmur, and the fluoroscopic view will show the pulsation in connection with the aorta.

Extremely Large Liver.

A large liver may reach the third rib; it may dislocate the heart to the left; it may move slightly by change of position of the patient. Here puncture by the needle will give pure blood, and an x-ray will show the supposed collection moving with the diaphragm (see Fig. 58).

(c) *Chronic Pleurisy*

Chronic pleurisy is the result of some previous infection of the pleura, the infection usually having taken place through the lung; it may be tuberculous—some writers declare that it is usually so. In post mortems one constantly encounters a lung firmly bound to the chest wall with more or less firm adhesions, the various lobes may be bound together, a collection of fluid either purulent or serous may be bound between the lobes or between the chest wall and a lobe. There may have been no physical signs or marked symptoms during life. If the adhesions are loose, there is not likely to be either physical signs or symptoms. If the union is quite firm between the parietal and visceral pleura there are the following physical signs: limitation of excursion of the affected side, impairment of the percussion note, the degree depending upon the thickness of the pleura and perhaps upon the actual lung expansion. There is also diminution of vocal fremitus and resonance. If the adhesions are old and very firm the neighboring organs will be *pulled toward* the affected side.

Conditions to be Differentiated from Chronic Pleurisy

This may be confounded with:

Liquid in the pleural cavity
Consolidation of the lung.

LIQUID IN THE PLEURAL CAVITY.

If the liquid is free in the pleural cavity the dullness is movable, but if the liquid is confined by adhesions or between the lobes of the lung, the dullness is stationary. Here the note is much duller than over a pleural thickening: there is a wooden sound to the note instead of a modified resonance. The neighboring organs are likely to be pushed away from the affected side.

A puncture with an aspiration exploratory needle will make a positive diagnosis provided the point of the needle actually reaches the liquid and the needle is large enough not to be plugged by a thick liquid. An x-ray carefully made by an expert should precede the use of an exploring needle whenever practicable.

CONSOLIDATION OF THE LUNG.

The diagnosis is not always easy from consolidation of the lung, but when a lung is consolidated, even though it be covered with a thickened pleura, there is a tendency to hear blowing breathing though the sound may be less loud than in the affected side. If the consolidation is acute, however, and there is no chronic thickening of the pleura, there is no difficulty in distinguishing the two. Here the voice and breath sounds and fremitus are increased, and the limitation of motion is, as a rule, less extensive than in chronic pleurisy.

11. Pneumothorax

Definition.—Pneumothorax is an accumulation of air within the pleural cavity.

Etiology.—It is usually sudden in its onset and may be the result of tuberculosis of the lungs, or of some other condition of the lung, or to a few causes other than diseases of the chest, as shown in the following list taken from Biach:

Causes of Pneumothorax in 914 Collected Cases

Tuberculosis	715 cases	Bronchiectasis	10 cases
Gangrene	65 "	Abscess of lung	10 "
Empyema	45 "	Emphysema	7 "
Traumatism	32 "	Infarct	4 "
Echinococcus	1 case	Perforated esophagus	2 "
Thoracentesis	1 "	Abscess bronchial glands	2 "
Worms	1 "	Fractured ribs	1 case
Ulcer of stomach	2 cases	Uncertain	14 cases
Peritonitis	1 case	Fractured sternum	1 case

In addition to the list above quoted, certain cases of so-called spontaneous pneumothorax occur which, so far as can be discovered, are not the result of tuberculosis, but are usually the result of a rupture of an emphysematous bleb in the lung. The local emphysema in these cases may surround an old tuberculous lesion. A liquid rarely forms in these spontaneous cases.

Symptoms.—The symptoms as stated are pain and dyspnea, usually abrupt in their onset. The affected chest usually gives a hyperresonant or tympanitic note on percussion. The adjacent organs—heart and lung—are usually dislocated, the heart being pushed to the left or right and the liver dislocated. Frequently in tuberculosis a liquid forms in the lower portion of the chest, and the case becomes one of pyopneumothorax.

In addition to hyperresonance and dislocation of organs usually suppressed breath and voice sounds are present, but at times there is amphoric breathing with metallic tingle, particularly at the end of the inspiration. If both liquid and air are present there is a marked metallic ring heard when a coin held against the side of the chest is struck lightly with another coin—"the coin sound." The voice sounds, usually suppressed, are frequently amphoric in character and if a liquid is present, a succussion splash can be heard. The affected side always measures more than does the unaffected. In rare instances there is dullness over the chest containing the air; here the dullness is due to great tension within the pleural cavity. X-ray examination gives much information. The side affected shows a perfectly clear space, while on the opposite side the normal lung

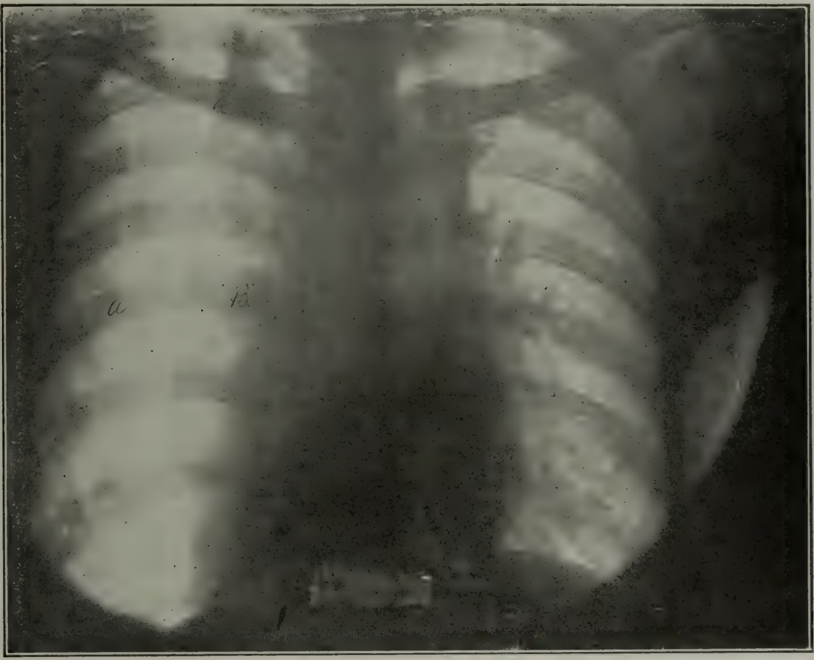


Fig. 66.—Pneumothorax. (a) Chest Filled with Air; (b) Collapsed Lung. (From Dr. H. K. Pancoast.)

gives the shadow normally seen. The collapsed lung shows at the apex (Fig. 66).

Conditions to be Differentiated from Pneumothorax

The condition may be mistaken for:
A diaphragmatic hernia
An extremely large cavity in the lung
An unusually extensive emphysema of the lung
A liquid collection within the pleural cavity
Subphrenic collection of pus
Pneumonia.

Diaphragmatic Hernia.

From diaphragmatic hernia the diagnosis is often difficult. Cases are on record where the entire chest on the affected side in a diaphragmatic hernia was hyperresonant. However, careful examination will reveal peristalsis over the area, and, furthermore, percussion over the abdomen will be transmitted very clearly to the chest. Then, too, there is not the same sudden onset; there is not the dislocation of organs to so great an extent as there is in pneumothorax; in addition, the hernia occurs on the left side of the chest and not on the right.

Extremely Large Cavity in the Lung.

A large pulmonary cavity has been mistaken for pneumothorax. It can be diagnosed by the history of a preceding tuberculosis with long-standing and increasing signs of cavity formation with expectoration of a large amount of liquid. The onset is not sudden as in pneumothorax.

Unusually Extensive Emphysema of the Lung.

An emphysema can at once be differentiated by the fact that it is practically always bilateral, whereas pneumothorax is of necessity always unilateral. Metallic tinkle, succussion splash, amphoric breathing or coin sound are not present.

Liquid Collection Within the Pleural Cavity.

The condition may be mistaken for a liquid collection when the intrathoracic tension due to the air is great because of the dullness over the collection of air. The very rapid appearance of the physical signs will help to make a diagnosis and the use of a needle will show the presence of air instead of liquid in the chest.

Subphrenic Collections of Pus.

A subphrenic collection of pus occurs occasionally, due to pus and air *below* the diaphragm, caused by the perforation of a viscus. This sometimes pushes up the diaphragm to such an extent that the signs indicate the collection to be in the chest. However, the symptoms are abdominal; the history is that of an abdominal condition; there is less cough; the heart is not so much dislocated as in pneumothorax. X-ray gives important evidence. The movement of the diaphragm will usually indicate the location—whether the collection is above or below the diaphragm.

Pneumonia.

In pneumonia and pleurisy with effusion, there may be a tympanitic note above the consolidated lung or the liquid, but the actual consolidation over the lung will designate pneumonia, and the absence of coin sound and the metallic tinkle over an effusion will distinguish the so-called Skoda phenomena from pneumothorax.

12. Empyema

(Pyothorax—Purulent Pleurisy)

Definition.—By empyema is meant a collection of pus in the pleural cavity, either free in the cavity or sacculated between the lung and the chest wall, between the diaphragm and the lung, or between the lobes of the lung.

Symptoms and Physical Sounds.—It has as its symptoms fever, leukocytosis, dyspnea and often palpitation of the heart. When the liquid is free in the pleural cavity, the physical signs differ but little from those present when the liquid is serum—there is the same enlargement of the affected side, dislocation of the heart and liver, movable dullness on percussion and loss of tactile fremitus, voice sounds and breath sounds.

When the collection is sacculated, and whether between the chest wall and the lung or between the lobes of the lung, the symptoms are the same. The physical signs are different, however. The dullness is not movable, indeed it may be a mere dullness and not flatness, because there may be a layer of compressed lung between the liquid and the chest wall, the liquid being either at the base of the lung or toward the mediastinum. The dullness is not as extensive as when the liquid is free. Over an area of greater or less extent, and usually in the line of one of the divisions between the lobes of the lung, there is more decided dullness, diminished or absent breath and voice sounds, with localized loss of tactile fremitus. An x-ray will show a decided shadow over these areas, be they great or small. The introduction of an aspirating needle will probably find the liquid and confirm the diagnosis. In cases in which there are good reasons for suspecting a liquid, an exploratory puncture is justifiable if the liquid cannot otherwise be found.

If the above symptoms and signs follow a pneumonia or if they be the first discovered, a purulent pleurisy may at once be suspected. Sometimes pus is expectorated as an indication of rupture of the purulent sac into the bronchus. Sometimes there is pulsation over the collection. Sailer has called attention to this in an exhaustive article.

Conditions to be Differentiated from Empyema

Serofibrinous effusion
Hydrothorax
Abscess of the lung
Bronchiectasis
Pneumonia
Tuberculosis
Tumors of the lung or pleura
Aneurism.

SEROFIBRINOUS EFFUSION.

Serofibrinous collection has less fever and less leukocytosis than empyema; there are less severe general symptoms. An x-ray will show the same shadow but a puncture by means of the aspiratory needle will show serum instead of pus. There is more apt to be egophony, which seldom occurs in purulent collections, although it may be present in children.

HYDROTHORAX.

Hydrothorax, a collection of fluid the result of cardiac disease, tumors or aneurism, can be diagnosticated by the absence of all of the symptoms of pleurisy and the presence of signs of cardiac disease, tumor or aneurism plus the pleural effusion. The aspirating needle will complete the diagnosis.

ABSCESS OF THE LUNG.

Abscess of the lung is difficult to differentiate. The condition more likely follows a general infection or a thrombus; if pus is expectorated the physical signs will largely disappear. An x-ray will show shadows in the lungs, often remote from the interlobar line.

BRONCHIECTASIS.

Bronchiectasis may cause confusion by some of the physical signs, particularly when an empyema has ruptured into a bronchus, but the condition is much less acute; there is no fever, no leukocytosis. The pus is often fetid in character.

PNEUMONIA.

Pneumonia is more acute; there is dullness, marked blowing breathing and increased fremitus. In certain cases, however, where the pleura is thickened for any reason, the physical signs are less marked than they are in frank cases. If a case of suspected pneumonia lasts over ten days or two weeks, especially if the fever is septic in type, careful and repeated examinations must be made to locate a liquid.

TUBERCULOSIS.

Tuberculosis may be suspected in cases following pneumonia, with aberrant signs, and in which a liquid cannot be demonstrated. However, unless tubercle bacilli can be discovered, or unequivocal signs of consolidation are found, an exploratory incision should be made.

Tuberculosis as a rule (though there are acute cases in abundance) is more chronic, wasting more marked and expectoration more profuse. As to the nature of the pus, tubercle bacilli may sometimes be demonstrated by using antiformin or a strong solution of alkalies, by boiling or allowing to stand in an oven, by washing and staining, or by animal inoculations made with small amount of the pus into the groins or subcutaneously into the animal.

TUMORS OF THE LUNG OR PLEURA.

New growths of the lung and pleura may give rise to exactly the same physical signs as a liquid; there is less fever, the cases are more chronic. One case reported by the writer of sarcoma of the pleura began as a sudden pneumothorax, eventuated in what appeared to be a liquid but incision demonstrated the growth, which at post mortem was found to occupy the whole pleural cavity. A puncture with a needle will not show liquid. An x-ray will be of value. Areas of consolidation are found in various parts of the lung.

13. Hemorrhagic Infarct of the Lung

Characteristic Features.—Hemorrhagic infarct of the lung is characterized by sudden pain in the side of the chest, dyspnea, faintness and usually hemoptysis.

Origin.—This condition is the result of an embolus in a bronchial vessel of greater or less size. The primary cause is usually a thrombus in some portion of the body, the veins of the legs, the uterus and auricles of the heart being the most frequent source. When it follows a surgical operation, the embolus has its origin in a thrombosis of a vessel which has been obliterated.

The greater the size of the pulmonary vessel occluded, the greater the amount of shock and hemorrhage. The physical signs and symptoms are due to the infarction resulting from the embolus. If the embolus is small the physical signs may be nil, but if any large area is involved near the surface of the lung there is dullness, a friction sound, blowing breathing and exaggeration of the breath sounds, or both may be less marked than normal.

Conditions to be Differentiated from Hemorrhagic Infarct

Pneumonia
Pulmonary hemorrhage of other origin
Pleurisy
Acute tuberculosis.

PNEUMONIA.

It is distinguished from pneumonia by the absence of a chill, high fever and leukocytosis, and also by the fact that it occurs suddenly in heart disease or the result of a phlebitis. There is no pulmonary hemorrhage in ordinary pneumonia and there is no shock.

Pulmonary Hemorrhage of Other Origin.

In pulmonary hemorrhage of other origin, in *tubercular hemorrhage* or in *hemorrhage from mitral stenosis* there is not the initial shock present in infarct and the hemorrhage is more severe. Also there is no pain in pulmonary hemorrhage due to tuberculosis, or in hemorrhage from mitral stenosis; in none of these is there any sign of a thrombus elsewhere in the body. But it must be remembered that a thrombus may be primary in an auricle, and this primary thrombus give no sign.

Pleurisy.

In pleurisy there is not the same shock which appears in hemorrhagic infarct and there is no hemorrhage.

Acute Tuberculosis.

Acute tuberculosis is often ushered in with severe symptoms and by hemorrhage; but there is no primary shock and there are no signs of a primary focus for the thrombus.

14. Mediastinal Disease

Mediastinal disease may take the form of a new growth. Almost all forms of tumor have been found in the mediastinum, but the benign are rare, the commonest being sarcoma, carcinoma, abscess, simple mediastinal cyst; an inflammation of the connective tissue in the mediastinum usually due to tuberculosis is also found.

Origin.—All of these conditions owe their symptoms particularly to the fact that pressure is made by encroaching upon the organs which pass in or near the mediastinum—the trachea, esophagus, or great vessels.

Diagnosis.—The differential diagnosis which is important from the standpoint of treatment is: (1) between a new growth and an abscess or simple mediastinitis, (2) between a mediastinal mass and an intratracheal or intralaryngeal obstruction, (3) between a growth or abscess and an aneurism.

A new growth is slow in its onset; it is usually unaccompanied by fever; there is rarely leukocytosis. There is wasting of the body in new growth, which is not likely to be so severe in abscess. The pain is usually less in new growth than in the latter. Finally an exploratory puncture of the mass will show pus in an abscess which of course is absent in a new growth.

A simple mediastinitis may give all of the obstructive signs of a new growth. There is the history however in all of these cases either of tuberculosis or syphilis. The writer has seen the great veins of the neck so completely obliterated in simple mediastinitis that there was much the same cyanosis of the face, neck and upper limbs such as often occurs from the pressure of a new growth or from an aneurism which ruptures into the vena cava.

The differentiating signs are the history of tuberculosis or syphilis in the mediastinitis, together with the absence of a visible mass or a very marked substernal dullness, and of pulsation which may occur in a new growth resting upon the arch of the aorta.

A new growth of the mediastinum often simulates *an aneurism.* Pulsation may be present, but it is always transmitted and never expansile. A diastolic shock is not present in a new growth, neither is a tracheal tug present, though one can conceive of adhesions between growth, aorta and trachea which will make such a pull upon the trachea and cause the downward pull.

Irregularity of the pupils, pulse, and recurrent laryngeal paralysis are very rare in mediastinal new growths and are extremely common in an aneurism. A bruit may be present over a growth or absent in an aneurism, though it is much more likely to be present in an aneurism. An x-ray picture, particularly a fluoroscopic examination, will show any pulsation which may be present to be expansile if the condition is due to aneurism.

Intralaryngeal growths or asthma might be mistaken for a mediastinal tumor. However, if the growth is in the larynx the dyspnea is more likely to be spasmodic; in new growth the dyspnea is continuous. In the laryngeal growth the use of a mirror will show the presence of a growth in the lumen of the tube. Physical examination of the upper chest will fail entirely to show any dullness, mass, or other signs of a new growth.

Asthma might possibly be mistaken for a new growth, but here the dyspnea is expiratory in character and there is entire absence of the high pitched note heard over the trachea in obstruction. Then there is the fact that certain cases of asthma are precipitated by close proximity to certain animals. There is no dullness under the sternum.

Section VII

Diseases of the Circulatory System

1. Palpitation of the Heart

Palpitation of the heart is a condition in which the patient is conscious of the beating of the heart.

Causes.—It is due to many causes. It may occur in individuals with diseased hearts or in those who have no organic change in the heart.

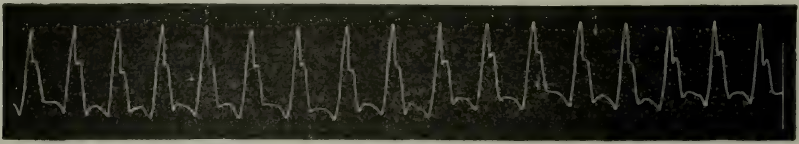


Fig. 67.—Tracing of Radial Pulse During Attack of Palpitation. (After Mackenzie.)

Palpitation occurring in individuals with normal hearts may be caused by excitement, by attacks of indigestion, or by the action of substances such as alcohol, tea, coffee or tobacco on the heart. It is common also in nervous individuals. Anything which startles the individual may cause an attack.

Symptoms.—There is frequently distress; the heart's action may be rapid but perfectly regular as seen in the cut from Mackenzie (Figs. 67 and 67A). At times the action is not rapid, but there is an uncomfortable feeling of oppression in the cardiac region. Sometimes there is extreme irregularity.

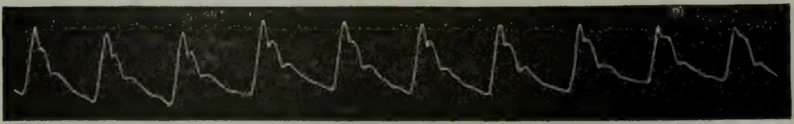


Fig. 67A.—Tracing from Same Individual as Fig. 67. Heart Beating Normally. (After Mackenzie.)

Palpitation also is one of the symptoms of diseased hearts, where the rapidity of action and the irregularity are the result of the stimulus to contraction, having either begun in an abnormal portion of the heart, or where there is difficulty of conduction, owing to disease of the bundle of His.

The important point is to distinguish a *palpitation* due to conditions outside of the heart from *a palpitation due to organic changes in the heart.*

Diagnosis.—A young individual whose heart beats rapidly, irregularly, or with only a slight abortive systole occasionally, the sounds being of proper character with no impairment of the muscular element, and in whom no murmur can be heard, is probably the subject of functional palpitation. Again, an individual who is past middle age, in whom there is irregularity of the heart's action, combined with rapidity, or one who has been the subject of an infection, and whose resisting power is lessened, and particularly if there be distinct interruption in the heart's rhythm, in the manner later to be taken up, is probably the subject of palpitation due to some organic change in the heart.

2. Sinus Irregularity

The normal rate of the adult heart is about 70-75 per minute, each beat following the other in regular sequence and with uniform force. This regular action of the heart is caused by stimulation of the so-called sino-auricular node. The node responds to this stimulation. The contraction is carried from the node and is transmitted through a bundle of tissue connecting the auricles and the ventricles—the so-called bundle of His—to the ventricles. The sino-auricular node is under the influence of the pneumogastric nerve.

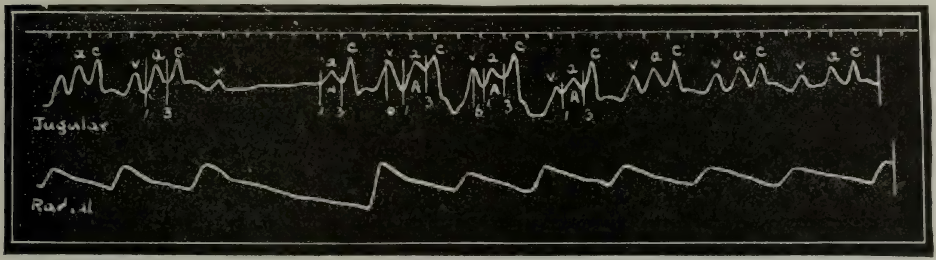


Fig. 68.—Sinus Irregularity, Occasional Slowing of Whole Heart. A-C Interval Is Not Affected by Variations in Rate. (After Mackenzie.)

Paralysis of the pneumogastric nerve can be brought about by atropin, which will cause an abnormal rapidity of the heart's action. Inhibition of the nerve will bring about slowing of the heart, because of its action on the node. The latter condition is seen in certain cases of acute fevers, jaundice, aortic stenosis and in certain cerebral conditions. It may appear in the course of apparent perfect health.

In some persons there is a marked irregularity of the heart during certain parts of the respiratory act. The heart beats fast during deep inspiration and slows during expiration. This is extremely common in children. It is typical of sinus irregularity and has no clinical significance. It is due to some disturbance of the pneumogastric nerve. Other forms of irregularity which owe their origin to stimulation or inhibition of the vagus, which have no relation to respiration, also occur. They are without danger to the individual, their chief importance being their effect upon the mind of the patient. Rapid or irregular heart action always alarms the patient; it is therefore important to differentiate the functional forms of irregularity from other and more serious irregularities of the heart.

In sinus irregularity, the auricular beats and the apex beats always correspond to the jugular and radial pulse, the tracing showing that the whole heart takes part in the irregularity. They can be recognized by the fact that they occur early in life, particularly before ten years of age and from the added facts that the sounds are normal in character and in force, that the reserve power of the heart is normal and that they disappear usually upon exercise. They can be unmistakably recognized by graphic tracings when such can be obtained, these showing that though the heart action is irregular in rhythm, the auricular contraction and the ventricular contraction follow each other in sequence (Fig. 69). Sinus irregularity is to be distinguished from the irregularities to be described later.

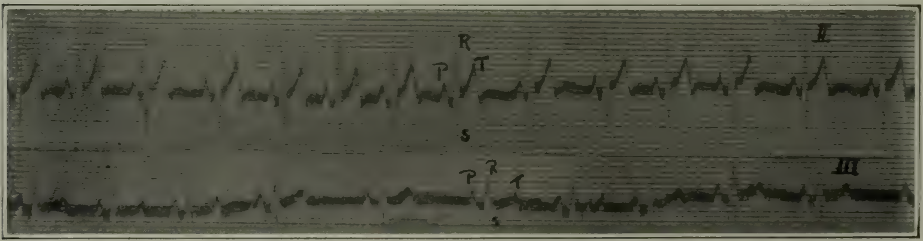


Fig. 69.—Sinus Arhythmia in a Young Adult. The Only Irregularity Is in the Disposition of the Beats Due to Varying Length of Diastoles. The Regular Sequence of Auricular and Ventricular Contractions Is Shown by the Constant Presence of P R S T in Each Beat. P Represents Auricular Contraction. R S T Represents Ventricular Contraction. (Kindness of Drs. Talley and Hewson.)

3. Premature Contractions

(*Extrasystole*)

Causes.—These irregularities are the result of stimuli arising from some portion of the heart muscle other than the sino-auricular node. They may arise in the auricle, in the bundle of His, or in the ventricle. The impulse which gives rise to a premature contraction is formed at a phenomenally rapid rate, and there is an absence of a tendency for the phenomenon to repeat itself (Lewis).

Occurrence.—The heart may be beating normally, when suddenly there is a premature impulse (extrasystole). If the abnormal stimulus comes from the ventricle the ventricle will rest after the premature contraction until an auricular impulse reaches it. If, on the other hand, the impulse come from an abnormal point in the auricle, both an auricular and ventricular impulse occurs and there is a disturbance of the normal rhythm indicated by a long pause, but the latter is not equal to two complete cycles of the normal rhythm.

These extrasystoles occur in various conditions. There may be organic cardiac disease, or they may occur where there is absence of organic

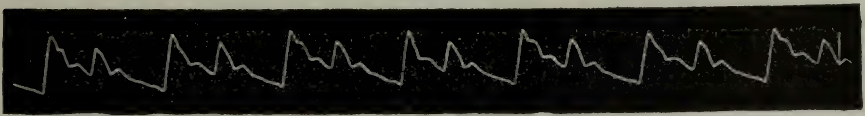


Fig. 70.—Pulsus bigeminus Due to an Extrasystole Occurring After Each Normal Beat. (After Mackenzie.)

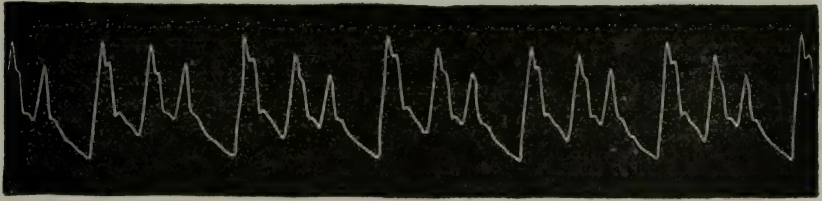


Fig. 71.—Extrasystole Occurring After Every Two Normal Beats. (After Mackenzie.)

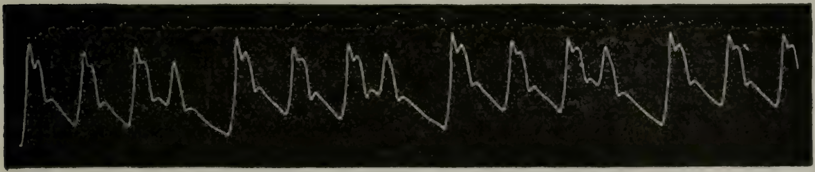


Fig. 72.—Extrasystole Occurring After Every Three Normal Beats. (After Mackenzie.)

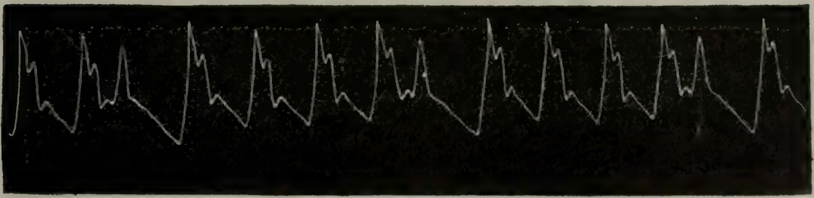


Fig. 73.—Extrasystole Occurring After Every Four Normal Beats. (After Mackenzie.)

heart disease. Premature contractions rarely occur in a rapidly beating heart.

Symptoms.—In certain cases there are no symptoms except the extra heart beat of which the patient may be unconscious. In other cases there is a sense of cardiac palpitation; there may be a tendency to syncope or a feeling of distress.

The regular sequence of sounds is interrupted by two short, sharp sounds followed by a long pause. If short sounds are heard at the apex during an interruption of the radial pulse, they are surely due to premature contraction. Lewis gives these evidences of the condition:

> "1. When a systole of a regularly beating ventricle is replaced by a premature beat, this abnormal contraction is accompanied by an early apex thrust and by (a) a weak arterial wave and two extra sounds which, together with those of the preceding rhythmic beat, form a group of four, or by an intermission in the arterial pulse and one extra sound, forming with the sounds of the preceding rhythmic beat, a group of three.
>
> "2. When each third beat of the regular ventricular rhythm is replaced by a premature beat, we find a grouping of the apex thrusts in threes, of which the third beat in each group is premature. The arterial beats (a) are grouped in threes, with groupings of the apical sounds, so that two normal heart sounds alternate with a group of four sounds, or (b) are paired with grouping of the apical sounds, so that two normal heart sounds alternate with a group of three sounds.
>
> "3. Premature beats which alternate with rhythmic beats give rise to pairing of the apical thrusts and to (a) pairing of arterial beats, of which the second stroke is weak, and to groupings of heart sounds in fours, or to (b) halving of the rate of the arterial pulse and heart sounds in groups of threes."

These various forms are seen in the following cuts from Mackenzie and electrocardiograms from Dr. James E. Talley (Figs. 74A, 74B, 74C).

Diagnosis.—The differentiation of premature auricular and ventricular beats is not always possible without full intrumental examination.

The effect of premature beats upon the auscultatory signs, when murmurs are present, are manifold; yet most of them can be foretold if the general principles are grasped. A systolic mitral murmur will be found with the premature as well as with the rhythmic beat, but it is usually short and may be absent. At the base of the heart in aortic disease, a systolic or diastolic murmur is present when the premature beat raises the aortic valves. On the other hand, in mitral stenosis a presystolic mitral murmur is absent whether the premature beat is auricular or ventricular, but in the former instance it is often replaced by a diastolic sound. The absence of the presystolic murmur in the case of the auricular beat is attributable either to weakness of the premature contraction or to its coincidence with the preceding ventricular systole. These premature contractions may have no prognostic worth.

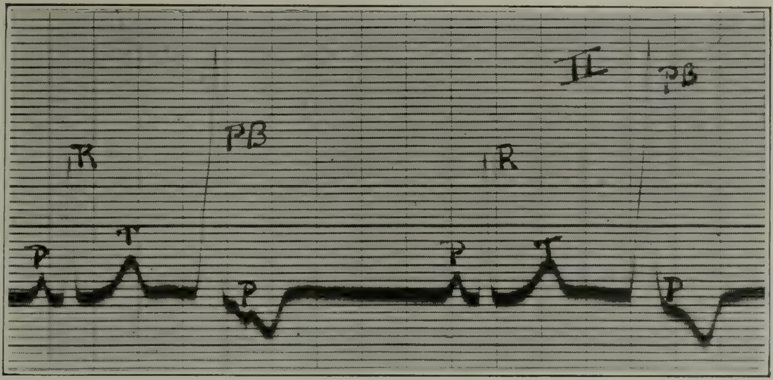


Fig. 74A.—Premature Ventricular Beats. Each Normal Beat, P R T, Is Followed by a Ventricular Premature Beat (P B) Arising in the Base of the Ventricle. The Auricular Beats (P's) Fall Regularly, But the Two Premature Beats Slightly Precede Two Normally Recurring P's. The Auricular Beats (P's) Cutting the Descending Limbs of the Premature Beats Give Rise to No Contraction Because the Ventricles Are Just Finishing Contractions, Hence Are in the "Refractory Phase." This Coupling of Beats Frequently Occurs Under Digitalis and Is a Sign for Withdrawal of the Drug. (Kindness of Drs. Jas. Talley and Hewson.)

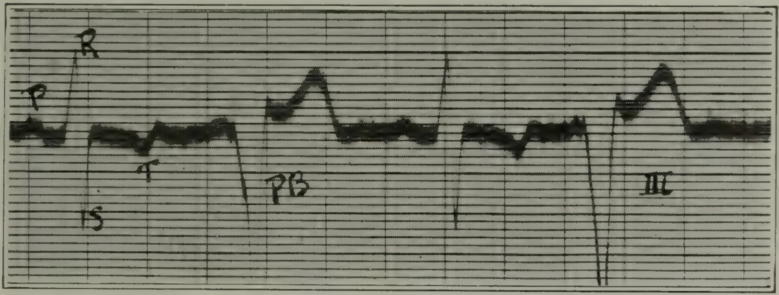


Fig. 74B.—Premature Ventricular Beats. Each Normal Beat, P R S T, Is Followed by a Ventricular Premature Beat (P B) Arising in the Apical Portion of the Ventricle. Note That T Is Inverted Instead of Being Normally Upright as in A. Such an Inverted T in Lead II in a Patient Not Under the Influence of Digitalis Has Been Considered a Bad Prognostic Sign. (Kindness of Drs. Jas. Talley and Hewson.)

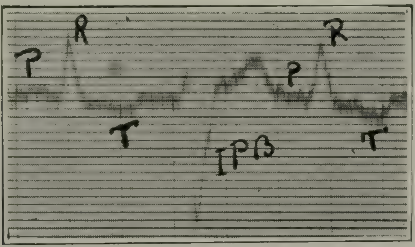
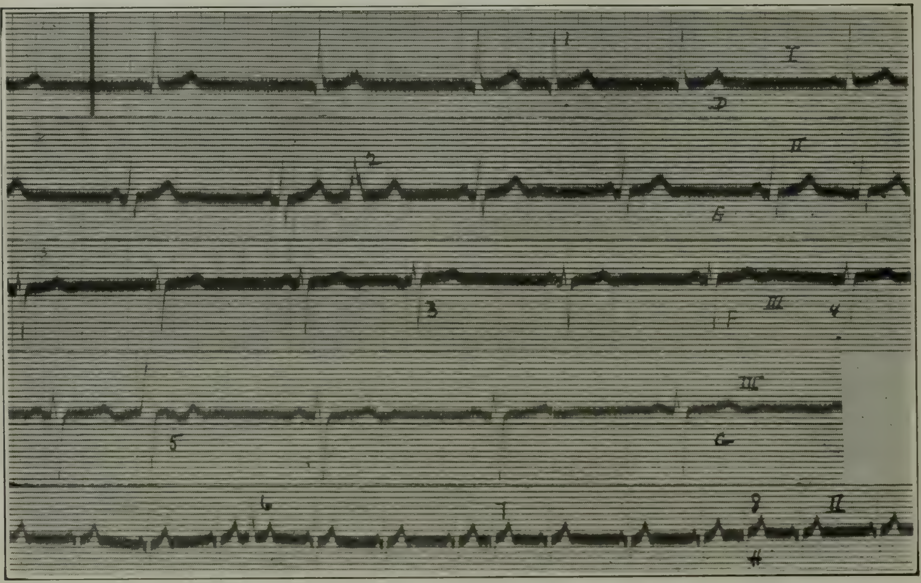


Fig. 74C.—Ventricular Premature Beats. The Ventricular Premature Beat Is an Occasional Event Interpolated Between Two Rhythmic Beats. (Kindness of Drs. Talley and Hewson.)

The extrasystole must be differentiated from sinus arhythmia and heart block.

Also the cases in which the extrasystole is an indication of a serious condition must be distinguished from those in which the phenomenon is of no serious importance.

In sinus rhythm, as already stated, the auricular and ventricular contractions follow each other in regular sequence. There are irregular ventricular contractions in cases of extrasystole. Sinus arhythmias occur most commonly in the young; premature contractions occur at any age. Sinus arhythmia is not indicative of any serious cardiac disease.



Figs. 74 D, E, F, G and H.—Auricular Premature Beats Are Taken from a Single Subject and Each Strip Shows One or More Auricular Premature Beats. D¹ Is Premature and Its Outline Conforms Exactly to the Type of the Regular Ventricular Beats. It Is Due to a Supraventricular Impulse Evidently Arising Near the Sino-auricular Node. P Is Poorly Developed Throughout the Curve.

E² and G⁵ Are Due to Auricular Impulses Which Slightly Notch T. The Abnormal Ventricular Responses Are Due to Deficient Conduction Along Certain Tracts of the Auriculoventricular Bundle Which Causes an Abnormal Distribution of the Impulse to the Ventricles. Such Contractions Are Called Aberrant.

F³ and ⁴ Are Auricular Premature Beats Due to Impulses Arising Not in the Pacemaker But at Another Focus in the Auricle. This Is Shown by the "Ectopic" P Preceding Them. This P Is a Downward Deflection While the P of the Regular Beats Is Upright.

H Should Be Compared with Fig. 69, Sinus Arhythmia. In the Latter Each Ventricular Contraction Is Preceded by a P of Normal Type, Whereas in H the Three Auricular Premature Beats, 6, 7, 8, Are Due to Small Ectopic P's Which Appear to Be Deflected Downward. (Kindness of Drs. Talley and Hewson.)

Heart block is recognized by the facts that there is a lengthened period between the auricular and ventricular beat, that the ventricular beat is lacking, or the auricle and the ventricle beat independently of each other.

Cases in which premature contractions are of serious import are those in which there are symptoms of dyspnea, palpitation, vertigo, etc.—the result of diminished cardiac reserve power.

4. Heart Block

Etiology.—This form of cardiac irregularity is due to a fault in the conducting power of the auriculoventricular bundle of His. It may be acute or chronic, and may be partial or complete. It is *partial* when the auricular impulse is merely delayed in passing through the auriculoventricular bundle, or when a particular impulse from the auricle fails to reach the ventricle. It is complete when the auricle and the ventricle beat independently of each other—they are completely disassociated.

Heart block may occur at any age; the cases of heart block caused by rheumatism are more common, according to Lewis, between 10 and 35 years of age simply because the diseases which produce the disturbance of the bundle are more common between those ages. It often occurs as the result of infectious diseases, rheumatism being the most common infection, followed by diphtheria, influenza, typhoid fever and pneumonia, in the order named. In the course of these infections heart block is acute and may be temporary, recovering as the temporary disturbance disappears.

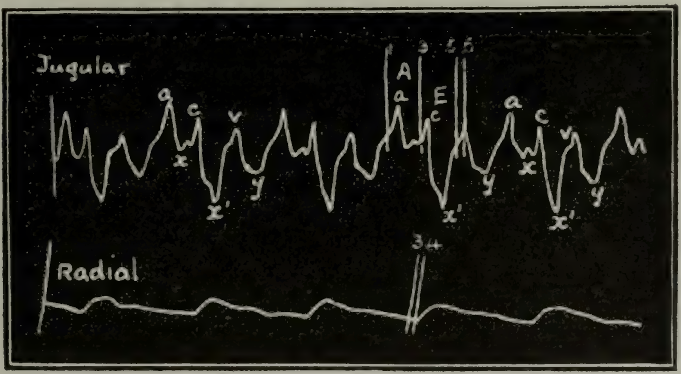


Fig. 75.—Heart Block. The Tracing Shows a Great Increase in the A-C Interval (Space A) Due to a Delay in the Stimulus Passing from Auricle to Ventricle. (After Mackenzie.)

Chronic heart block is caused by the permanent changes in the auriculoventricular bundle resulting from the above named diseases, and especially from rheumatism, syphilis, arteriosclerosis and fatty degeneration. Many of the cases in this group occur in elderly individuals.

Heart block also occurs as a result of an overadministration of digitalis. There may possibly be some fault in the bundle itself in these cases, but the fault becomes much more pronounced by the use of the drug, owing to its effect upon the vagus.

Diagnosis.—It can best be recognized by the use of instruments of precision, such as the polygraph and electrocardiograph. Perhaps in the slighter grades of disturbance this is the only method by which it may be recognized. However, heart block may frequently be distinguished without the use of graphic tracings (Figs. 75, 76A and 76B).

The clinical points of value, and really the important points, are as follows: the apex beat is slower than the auricular which can be seen or felt in the jugular vein; again, according to Lewis, another means of diagnosis without a tracing is that obtained by auscultation. There is a muffled sound originating in the auricle, which can be heard when the A-V interval is widened, the latter always occurring to a greater or less degree when there is heart block. This gives rise either to a double first sound when the widening of the interval is slight, or a double second sound when the interval is greater. There is also a sound occurring in mitral stenosis when the pulse is regular; these are apical thrills and presystolic murmurs confined to mid-diastole.

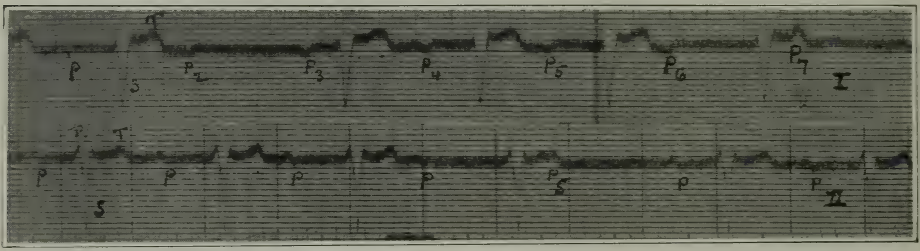


Fig. 76A.—Partial Heart Block—Lead I. Note the Continually Increasing Length of the P-R Interval After P^3, P^4, P^5, and P^6. P^2 and P^7 Fail to Provoke Any Ventricular Response, Thus Showing One Cause of Dropped Beats. The Same Condition Occurs in Lead II, the P-R Interval Varies and the P^5 Fails to Produce Any Ventricular Response. The Auricular Beats Recur Regularly But the Changing Length of the P-R Interval and the Dropping of a Beat After Each Fifth Auricular Contraction Produce Irregular Ventricular Contractions. (Kindness of Drs. Talley and Hewson.)

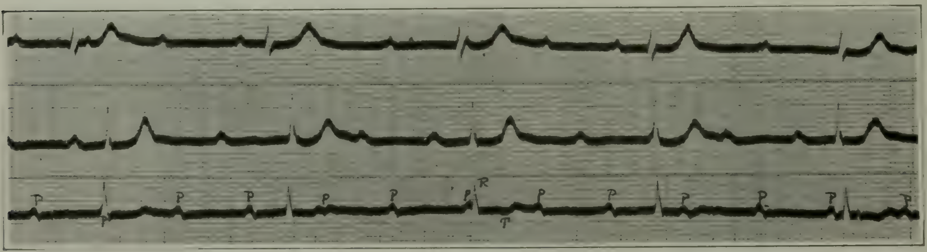


Fig. 76B.—Complete Heart Block. There Is Complete Dissociation: the Ventricular Rate Is Approximately 38, the Auricular Approximately 98. Auricle and Ventricle Each Beat Regularly But at Different Rates. The Auricular Contraction Falls Often Clear in Ventricular Diastole, Sometimes with R and Sometimes with T. (Kindness of Dr. Alfred E. Cohn.)

Heart block may be suspected always when the ventricle beats regularly and not more than 30 to the minute, and also when there is a single dropped beat at the wrist and the apex beat is neither felt nor heard during that interval. This latter is due to the failure of the ventricle to respond to the stimulus of the auricle and is therefore one form of heart block. The symptoms which occur have not only to do with the fact of disturbance in the bundle but are really much more concerned with injury to other portions of the heart. There is usually loss of reserve power, sometimes dyspnea, palpitation and vertigo; sometimes there are no symptoms whatever save a lack of reserve power and the irregular heart action. High systolic blood pressure in the presence of missed beats is a fairly good sign of the fair condition of the heart muscle.

In extreme cases of complete heart block the so-called Stokes-Adams syndrome sometimes occurs. This syndrome is the result of cerebral anemia. Here the auricle continues to beat and the ventricle either beats extremely slowly, 5 or 6 to the minute, or stands absolutely still; when the latter occurs, an epileptic form of convulsions appears. In some of these cases, the patients are more or less subject to vertigo, dimness of vision and hallucinations in the absence of the complete syndrome.

Heart block is to be differentiated from sinus irregularity and from cerebral conditions due to other causes, such as tumors of the brain and epilepsy. In sinus irregularity, as above stated, the whole heart takes part in the arhythmia. There is no disassociation between the ventricular and auricular rhythm: it occurs early in life; there are no signs of cardiac inefficiency.

Epilepsy and cerebral conditions such as tumors give rise to convulsions. But here while the heart action may be slow, it is usually regular and there is no disassociation between the ventricular and auricular beat. Then there are other signs of brain lesion, such as paralysis in tumors and a long-continued history of convulsions in epilepsy. Epilepsy, too, usually begins in early life, while Stokes-Adams syndromes occur much later.

5. Fibrillation of the Auricle

Etiology.—This important form of irregular heart beat is recognized by a total irregularity of the apex beat and of the radial pulse. The rate is extremely rapid. The systolic impulses follow each other without any regular sequence; they are now strong, now weak; they sometimes follow each other in rapid succession, sometimes more slowly. The tracing shows rapidly recurring curves of different heights—of different rhythm.

The condition is due to the fact that the auricle has entirely lost its systolic power, but has taken on a fibrillary tremor. In consequence of this, the ventricle is subjected to a series of stimulations at varying rates and of varying strength. The polygraph tracing shows without exception that the tracing of the jugular impulse is *systolic* and not diastolic (ventricular venous pulse).

Symptoms.—The symptoms of the condition are those of cardiac failure, sometimes appearing suddenly and sometimes gradually increasing in severity. These symptoms are breathlessness, vertigo, syncope, cough, edema, with very rapid and irregular cardiac impulse and radial pulse. There are cases of fibrillation without these serious symptoms, in which the pulse rate is much slower, but they can be best discovered by the use of the polygraph or the electrocardiograph.

When fibrillation of the auricle occurs in cases of mitral stenosis, the presystolic murmur disappears, to be replaced by a murmur which is diastolic and is best heard in the region of the apex. This is often mistaken for the murmur of aortic regurgitation.

In auricular fibrillation there is, during decompensation, a pulse deficiency; that is, the apical rate exceeds the radial pulse rate by a number of beats—in some cases there is 25 per cent deficiency. This is one of the cardinal signs of this disorder; it differentiates it from other cardiac lesions.

It has been shown that the cases of auricular fibrillation are most apt to occur in cases in which the heart is damaged as the result of rheumatism, and Mackenzie believes that it is in this type of irregularity that digitalis administration is by far of the greatest value. The whole heart is the seat of myocardial degeneration. The condition is to be distinguished from all other types of cardiac irregularity.

Diagnosis.—The conditions can always be differentiated by the use of a polygram or electrocardiogram (Figs. 77, 78A, 78B and 78C), when it will be seen that the jugular wave is systolic; also that the heart beats are continuously rapid and irregular in force and rhythm.

It occurs at any age. The diastolic murmur spoken of above, which occurs in cases of mitral stenosis, in a patient the subject of auricular fibrillation, can be distinguished from a murmur of aortic regurgitation by the fact that the murmur is heard only at the apex, while that of aortic regurgitation is heard at the base, and is conducted down the sternum and often heard best at either the third left intercostal space or at the xyphoid. There is in aortic regurgitation the characteristic pulse tracing, the hypertrophied heart and the capillary pulse which are wanting in cases of auricular fibrillation in mitral stenosis. Sinus rhythm occurs most commonly in the young, as above stated, and there the whole cardiac cycle is involved. Extrasystoles are interpolated beats between normal ones, and may sometimes be made to disappear by allowing the individual to exercise. This is not the fact in auricular fibrillation. In auricular fibrillation exercise will increase the irregularity. In paroxysmal tachycardia, the beats are usually perfectly regular but extremely rapid, the cases beginning suddenly and ending suddenly.

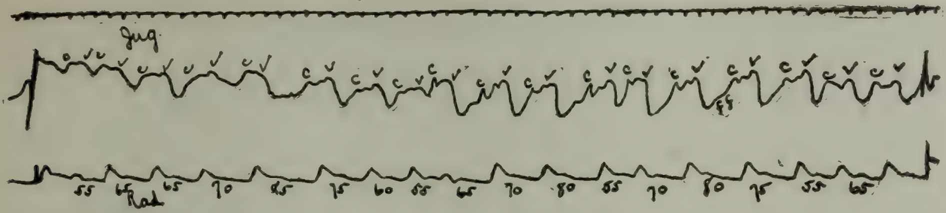


Fig. 77.—Polygraph Tracing of Auricular Fibrillation. Note the Varying Rate of the Radial Beats. The Radial Curve Will Not Space. The Jugular Tracing Shows the Ventricular Form of the Venous Pulse. (Kindness of Drs. Talley and Hewson.)

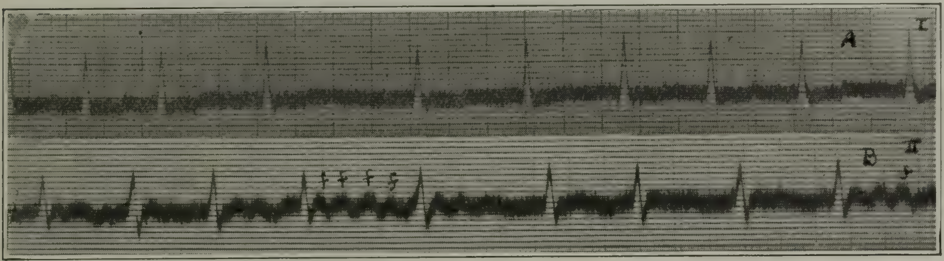


Fig. 78.—Auricular Fibrillation. A and B Are from Two Fibrillating Hearts. In A Note the Irregular Disposition of Beats, also the Lack of Any P Variation Before R, and the Irregularity in the Heights of R. There Is No Relationship Between the Height of R and the Length of Pause Preceding it.

In B There Is in Addition at Times the Oscillations (ff) Due to the Fibrillating Waves in the Auricle. (Kindness of Drs. Talley and Hewson.)

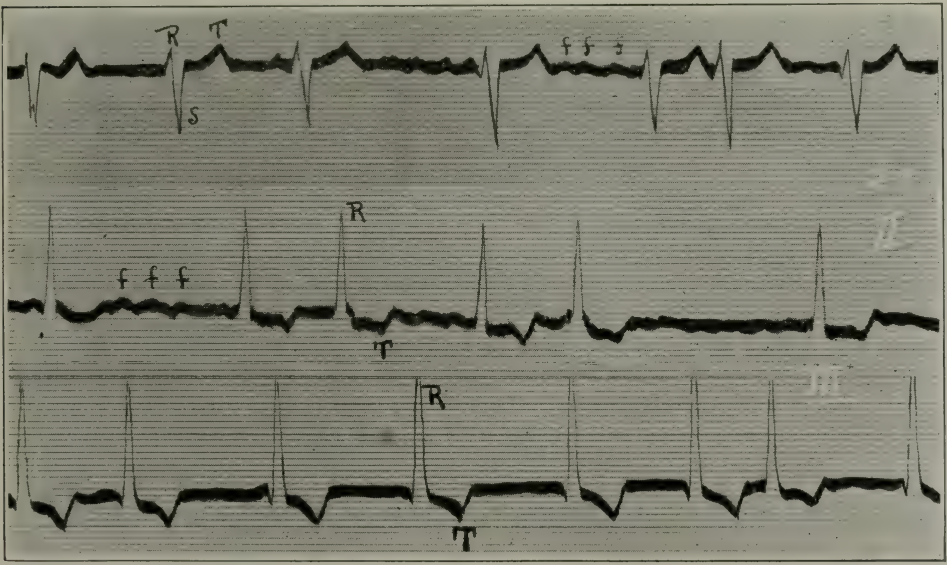


Fig. 78C.—Auricular Fibrillation. C Represents Three Leads from a Case of Auricular Fibrillation Under the Influence of Digitalis. The Beats Are Irregularly Placed; There Is No P Variation Preceding the Ventricular Beat Anywhere, the Height of R Bears No Relation to the Length of the Pause Preceding It. In This Subject T Is Inverted in Lead II. The Oscillations (ff) Are Shown. The Deep S in Lead I and the Tallest R in Lead III Show That the Heart Had a Preponderance of Muscle on the Right Side. (Kindness of Drs. Talley and Hewson.)

6. Paroxysmal Tachycardia

(*Delirium cordis*)

This is a condition of extremely rapid heart action—160 to 200 per minute—coming on suddenly and ending in the same manner, with or without apparent cardiac cause (Fig. 79). The rapid action is not affected by the position of the patient. It is due to the rapid succession of new and rhythmic pathological impulses.

Occurrence.—The attack may be only temporary or it may last a few hours or even months. It occurs at all ages, as early as two years and nine months, being most common between thirty and sixty years of age. The condition is more common in men. Many of the cases give no evidence of valvular or other lesions, but attacks do occur in the course of actual lesions, mitral stenosis being the most common.

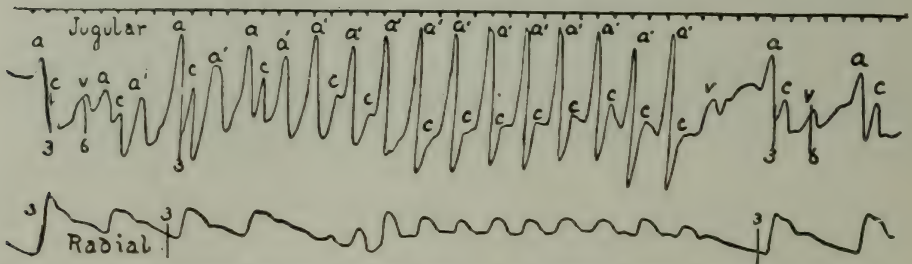


Fig. 79.—Simultaneous Tracings of the Jugular and Radial Pulses During One Attack of Paroxysmal Tachycardia. The First Cardiac Cycle in the Jugular Tracing Shows the Normal Events (*a*, *c*, *v*). The Second Cycle Shows the Normal Waves *a* and *c*, but the Wave Following Marked *a'* Occurs Earlier than the Wave *v* in the Previous Cycle, and is Due to Premature or Auricular Extrasystole, but Is Not Followed by a *c* Wave or by a Radial Pulse Beat. The Next Two Normal *a*, *c* Waves Are Each Followed by an Auricular Extrasystole (*a'*) with No Ventricular Response, as Shown by the Absence of the *c* Wave and the Radial Pulse Beat. These Are All "Interpolated Auricular Extrasystoles." After These There Follows a Series of Auricular Premature Beats (*a'*) to Which the Ventricle Responds as Shown by the *c* Waves and the Small Radial Pulse Beats.

The Onset of the Paroxysm Always Coincides with Great Distention of the Jugular Veins, Which Is Shown in the Tracing by the Greater Amplitude of the Auricular Waves *a'*. (After Mackenzie.)

The author has notes of one typical case in which absolutely no heart lesion could be diagnosticated in the beginning of the attacks, but which lately has developed a mitral stenosis. Another case, a man with typical attacks, showed only a slight breathlessness between these recurrences. Nothing was found at autopsy after several years of these paroxysms. The literature shows that there is usually some fibrosis of the cardiac muscle.

Overexertion or overexcitement may precipitate an attack, or it may occur without any cause which is apparent.

The Attack.—It begins without premonition and lasts a few minutes or hours. Sometimes the patient is much distressed, sometimes there is

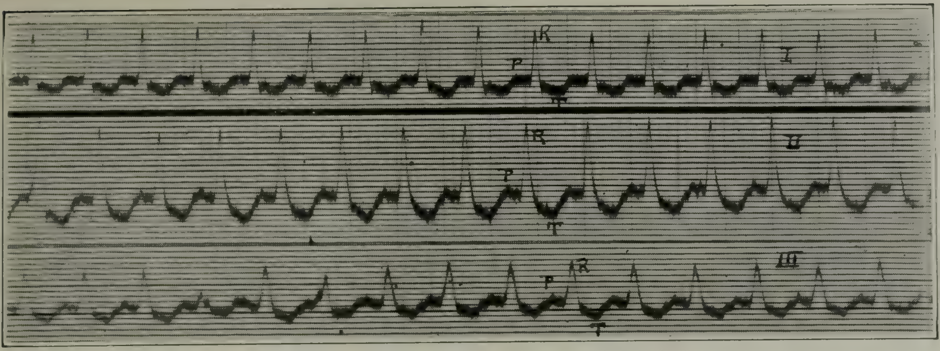


Fig. 80A.—Paroxysmal Tachycardia—Rapid Rate. Represents the Three Leads Taken During the Attack of Rapid Heart Action. T Is Inverted in All Curves Taken from This Patient. (Kindness of Drs. Talley and Hewson.)

no disturbance apart from the rapid heart action; there is often visible pulsation of the veins of the neck. The presystolic murmur is often not heard when these attacks occur in cases affected with mitral stenosis.

In a few minutes following the cessation of the paroxysm the patient feels perfectly well, provided the attack has not been accompanied by signs of cardiac decompensation. Sometimes nausea and vomiting occur; at times cardiac dilatation, cyanosis and expectoration of bloody froth are prominent. The pulse tracing shows very rapid heart beat; they are usually regular, and there is no sign of auricular fibrillations.

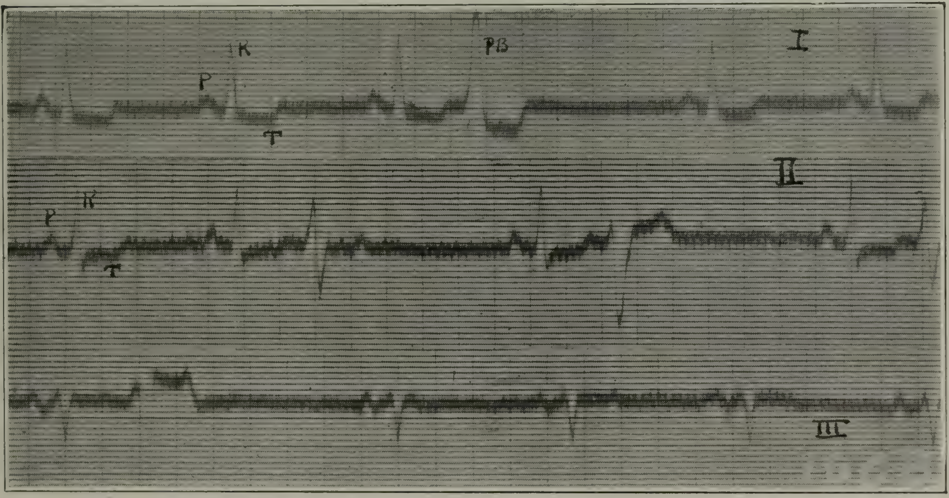


Fig. 80B.—Paroxysmal Tachycardia After Digitalis. B Was Taken After Administration of Digitalis. In Lead I the Regular Rhythm Is Intercepted by One Ventricular Premature Beat of the Basal Type. In Lead III There Is One Ventricular Premature Beat of the Apical Type. Definite Coupling Is Shown in Lead II. In the First and Third Pairs of Coupled Beats the Abnormal Beats Arise in the Auricle But Pursue an Abnormal Course in the Auriculoventricular Bundle Producing Aberrant Contractions. In the Second Pair of Coupled Beats the Coupling Is Due to a Ventricular Premature Beat Arising Toward the Apex. The Rapid Vibrations Shown Throughout the Curves Are Due to Muscular Tremors. (Kindness of Drs. Talley and Hewson.)

Differentiation.—The condition must be distinguished from:

A MERE RAPID HEART ACTION DUE TO EXCITEMENT, OVERSTRAIN OR AN ORGANIC DEFECT.

In the rapid heart which occurs in hysteria or in any nervous individual there is not the same sudden beginning and sudden ending as in this condition; there is no lesion of the heart.

The rapid heart which accompanies organic lesions is usually favorably affected by rest in a recumbent position. If there is fibrillation of the auricle the tracing will show a ventricular pulse in the jugular vein.

7. Auricular Flutter

Lewis, in his "Clinical Disorders of the Heart Beat," defines this condition as one in which the normal beats of the auricle are submerged by contractions of this chamber in response to a series of new, rhythmic and pathological impulses, varying in rate from 200 to 500 per minute.

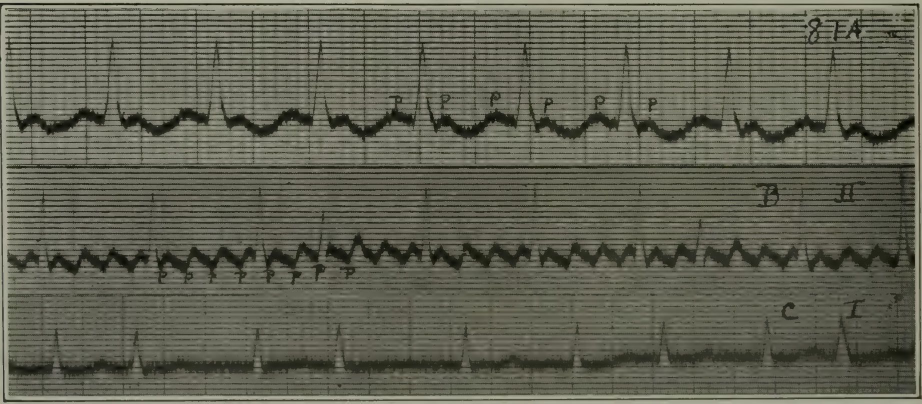


Fig. 81.—Auricular Flutter. The Three Curves Are from a Single Subject. A Represents the Condition When It Came Under Observation Uninfluenced by Any Drug. There Is 2:1 Heart Block. The Auricular Rate Is 320, the Ventricular Rate 160.
B Shows the Condition After Digitalis Was Given: Here There Is 3:1 and 4:1 Heart Block. The Auricular Rate Is 330. The Ventricular Rate 90.
C Was Taken After Further Administration of Digitalis Had Provoked Auricular Fibrillation. The Ventricular Rate Is 135. Though Digitalis Was Pushed to Coupling of Beats a Number of Times There Was No Return to Normal Rhythm. The Heart Has Continued to Fibrillate for Nearly a Year. The Patient Is a Man of 76 with No Valvular Lesion, Though He Had Inflammatory Rheumatism in His Youth. He Is Up and Around But with a Very Limited Field of Cardiac Response. (Kindness of Drs. Talley and Hewson.)

Diagnosis.—The condition is certainly recognized by the electrocardiogram (Fig. 81) and often this is the only positive means of making a diagnosis.

Symptoms.—In rare instances the condition may be recognized, when no cardiogram can be obtained, by *seeing* the extremely rapid action in the jugular veins.

Other evidences of flutter are those of ordinary cardiac weakness, such as shortness of breath, and in many cases cerebral symptoms, such as vertigo. Sometimes there are very few symptoms, even in cases lasting over periods of one year. The condition may last many years, or according to Mackenzie, it may appear as a terminal condition in certain diseases of the heart.

There is distinct limitation of the reserve power of the heart. Usually there is heart block of often a 2 to 1 rhythm, the auricle beating 320 per minute, and the ventricle 160.

A frequent symptom is Cheyne-Stokes respiration.

Mackenzie now believes that cases of paroxysmal tachycardia with a regular rhythm are due to auricular flutter: "If tachycardia persists for a month or more and is not changed in rate by change in posture, rest or exercise, the case is almost surely one of flutter."

Differentiation.—Flutter may be confused both with AURICULAR FIBRILLATION and PAROXYSMAL TACHYCARDIA. As stated above, Mackenzie believes that *prolonged cases of paroxysmal tachycardia are really flutter.* In auricular fibrillation the marked response to digitalis medication marks the difference from the ordinary case of flutter, which is irregularly affected by digitalis.

8. Alternation of the Pulse

(*Pulsus alternans*)

This is a condition in which, while the rhythm of the pulse is normal, alternate beats are much stronger than others. (Fig. 82.) It can be more satisfactorily distinguished by a tracing than by the finger, but some cases can be discerned by palpation of the radial artery.

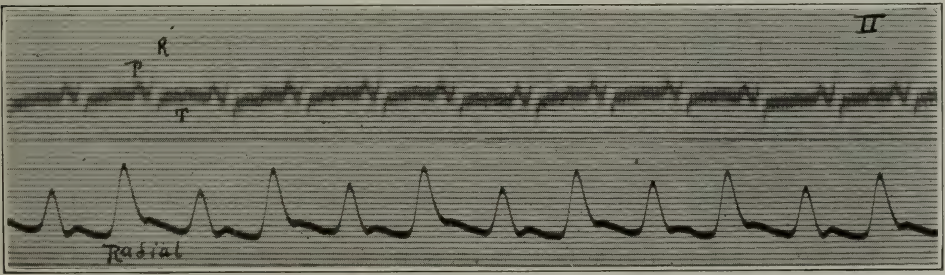


Fig. 82.—Alternation of the Pulse. This Figure Illustrates Pulsus alternans or Alternation of the Pulse. The Figure Gives Simultaneous Electrocardiograph and Polygraph Curves. As Usual the Polygraph Shows the Condition Better. The Inverted T in Lead II of the Electrocardiograph Curve, Though Not So Well Shown Here, Has Been Constant in Curves from This Patient. This Finding, Especially When Coupled with the Development of Alternation, Is Considered of Bad Prognostic Significance. (Kindness of Drs. Talley and Hewson.)

Occurrence.—The condition occurs in cases of acute illness in which the heart muscle is overtaxed or weakened; it occurs in individuals who are the subject of nephritis, of high blood pressure, of angina pectoris or of paroxysmal cardiac asthma. The prognosis of this character of pulse is grave.

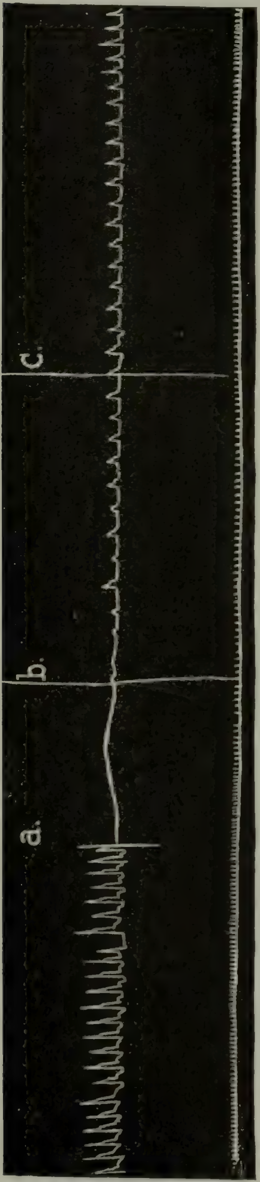


Fig. 83.—Alternation of Pulse Shown Between b and c. Made from Radial when the Brachial Is Compressed by Sphygmomanometer Cuff. (Kindness of Dr. Robert Pittfield.)

Treatment.—Every physician knows that in cases of acute illness, when the pulse begins to assume this rhythm, the heart muscle is beginning to flag under its load. Unfortunately for prognosis, the majority of instances cannot be recognized without the aid of a tracing. A simple polygraph tracing of the radial pulse is usually sufficient. In all cases where it persists, in the chronic as well as the acute conditions, it should be recognized that the heart is beginning to feel the strain and that immediate measures must be taken to relieve the condition. If it is possible to remove the cause, this must be done. If the cause itself cannot be removed, then the patient must be given all the relief possible through rest and other measures.

Hay of Liverpool, Herrick of Chicago, and Pittfield of Philadelphia, independently found that by occluding the radial pulse with the sphygmanometer, and then by slowly releasing the air, the strong heart beats will appear at the wrist and can be felt while the weak ones will not be felt. As the pressure is released further, the weak ones appear. At first the pulse is halved. The tracing in Fig. 83 shows this very well.

Differentiation.—Alternation of the pulse might be mistaken for a DICROTIC PULSE, but the irregularity is due to difference in the force of the heart beat and is not due to a double dicrotic wave. In dicrotism there is a double diastolic wave. It might also be mistaken for EXTRASYSTOLE, but here there are two ventricular beats. In the alternation of the pulse the tracing shows one beat weaker than another.

9. Pericarditis

(*a*) *Acute Fibrous Pericarditis*

Cause.—Acute pericarditis is the *result of an infection.* The infective organism is usually that of rheumatism, although the condition may occur in the course of pneumonia, scarlet fever and other infectious conditions.

Symptoms and Physical Signs.—The general symptoms are those of any acute infection. The disease is characterized by pain in the cardiac region; the pain sometimes is transferred to the abdomen, particularly to the region of the appendix. Depending upon the degree of inflammation, sometimes there is tumultuous action of the heart, dyspnea, cyanosis and difficulty in swallowing. Frequently the symptoms are so slight that only physical examination will allow of or even suggest a diagnosis. On examination by the stethoscope the heart's action is found to be increased in rapidity, sometimes altered in rhythm and accompanied by a to-and-fro friction rub. The heart's dullness is rarely increased in extent. If an effusion takes place, however, the heart dullness then increases in extent—sometimes the increase is very rapid. The dullness is of a peculiar form indicative of effusion, that is, with the apex up and the base below. The murmurs disappear, the heart sounds become distant and sometimes feeble and the action of the heart may be extremely irregular. The apex of the heart is frequently lifted to the interspace above. However, there are many instances where the heart is adherent to the pericardium in front and the effusion occurs posteriorly, often resembling a pleural effusion; the precordia bulges, especially in young individuals. The friction frequently recurs when the effusion disappears.

Conditions to be Differentiated from Acute Pericarditis

Endocarditis
Painful conditions of the chest other than pericarditis
Appendicitis
Dilatation of the heart
Pleural effusion.

ENDOCARDITIS.

From endocarditis the diagnosis must be made largely upon auscultation; the murmurs present in endocarditis are blowing in character and situated over the one or other orifice and are conducted in the direction of the blood streams. The pericardial murmur is not blowing, but is to-and-fro in character and appears to be close to the ear. Then the murmur of pericarditis usually disappears with the improved condition of the acute infection, whereas that of endocarditis remains because of the permanent injury done to the valve itself. Again the heart dullness in endocarditis suddenly increases only when there is marked cardiac decompensation, the result of the myocardial change which often accompanies endocarditis.

Painful Conditions of the Chest Other Than Pericarditis.

Pneumonia might be mistaken for pericarditis because of the transference of the pleuritic pain of pneumonia to the region of the heart, but the latter is characterized by an entirely different set of symptoms—consolidation of the lung, blowing breathing, rusty expectoration and pleurisy. It must never be forgotten, however, that pneumonia and pericarditis may occur simultaneously in the same individual.

Appendicitis.

Pericarditis might be mistaken for appendicitis by the transference of the pain to the iliac fossa. The writer has seen pericarditis mistaken for appendicitis, but the mistake rarely occurs in the reverse way. In appendicitis, however, careful examination will certainly show tenderness over the McBurney point; there will be no disturbance of the heart rhythm and no murmur over the cardiac region, unless it be an old murmur.

Dilatation of the Heart.

Dilatation of the heart is marked by cardiac decompensation earlier in the course of the condition than it is in pericarditis. Marked pericardial inflammation and effusion may occur without cyanosis, edema or breathlessness, though if the case advances those signs sooner or later make their appearance. In dilatation, the cardiac impulse is diffuse and often felt over the whole area of dullness. In pericardial effusion the impulse is difficult to hear and is often limited to the upper area of dullness, and this latter is not pyriform in outline as it is in effusion. The sounds are more readily heard than in effusion; there is usually an endocardial murmur which can be heard and which rarely disappears entirely with the increase of cardiac dullness.

When a pericarditis is accompanied by a severe myocarditis so that a dilatation occurs, then it is almost impossible to make a differential diagnosis. And when a heart either by adhesion or for some other reason is retained in the front of the chest, the condition of effusion may readily be mistaken for dilatation.

Pleural Effusion.

Left pleural effusion may be mistaken for pericardial effusion, or vice versa. In pleural effusion, however, the heart is pushed to the right and the area of dullness extends far beyond the area of cardiac impulse. Sometimes, on the other hand, large pericardial effusions have been mistaken for pleural effusion. As to whether a pericardial effusion is purulent, serous, or hemorrhagic, paracentesis is the only positive means of deciding; increasing leukocytosis and a septic fever indicate that the case is purulent in type.

(*b*) *Chronic Adhesive Pericarditis*

Diagnosis.—Adhesive pericarditis may be so free of symptoms that only a routine examination will show the presence of the disease. At other times there are signs of failing cardiac compensation which confuse and lead to the diagnosis of chronic endocarditis; sometimes there is pain which may readily be mistaken for angina pectoris.

The important points are: a history of an antecedent acute pericarditis, failing compensation and pain; retraction of the apex beat is perhaps the most important sign. With every systole the region or neighborhood of the apex of the heart is retracted. Systolic retraction of the left, tenth and eleventh interspace posteriorly (Broadbent's sign) is important. A friction may be felt over the heart. Collapse of the veins of the neck is also an important sign.

On auscultation, weak heart sounds accompanied by a friction murmur may be heard. Sometimes a loud systolic murmur may be heard either at the apex or xyphoid, the result of relative dilatation of the heart.

Conditions to be Differentiated from Chronic Pericarditis

The cases can be mistaken for:

Dilatation of the heart due to endocarditis
Angina pectoris
Primary cirrhosis of the liver.

DILATATION OF THE HEART DUE TO ENDOCARDITIS.

Dilatation of the heart without pericarditis is not accompanied by a friction sound; there is more likely to be edema, cyanosis and palpitation. The murmurs present are distinctly due to insufficiency or stenosis of one or the other orifice. There should be no difficulty in making a diagnosis from the stenotic murmurs. In aortic regurgitation the character of the murmurs, the shape of the cardiac dullness and the capillary pulse and Corrigan's pulse in aortic regurgitation should make the matter clear.

ANGINA PECTORIS.

Angina pectoris is simulated by pain, but in true angina pectoris due to cardiac sclerosis there is no pericardial friction rub. In the pain of adhesive pericarditis there is not the same conduction of the pain as there is in true angina. Pericarditis rarely, if ever, causes sudden death.

The dilatation of the heart due to high blood pressure and renal disease can be differentiated by the presence of these conditions and the absence of a friction rub and systolic retraction.

(*c*) *Pericardial Effusion*

This condition, as indicated above, may result from the formation of a liquid exudate following the inflammation of the two layers of the pericardium, when it is really a part of the acute pericarditis. The effusion instead of being an inflammatory exudate may be a transudate, the result of chronic decompensation, or of chronic nephritis.

A diagnosis is less difficult when the exudate is the result of inflammation than when it is a transudate, for then one has the previous history of pain, fever and friction sound to guide him.

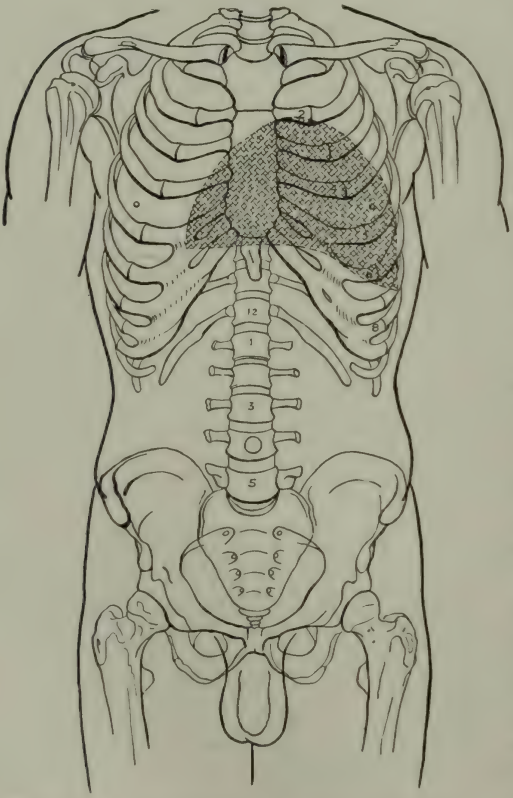


Fig. 84.—Area of Dullness in Pericardial Effusion. (Observed at St. Timothy's Hospital.)

Physical Signs.—The physical signs present in the acute variety are: disappearance of the friction sound with gradual enlargement of the cardiac area of dullness which assumes a pyriform shape, disappearance or lifting of the apex beat, bulging of the pericardium (Fig. 84), muffling of the heart sounds, an abnormal dullness to the right of the sternum (Rotch's sign) not due to dilatation or dislocation of the heart, and sometimes signs of cardiac distress.

A large pericardial effusion gives a dullness in the lower portion of the chest, either occupying the whole chest or an area of dullness at the lower angle of the scapula posteriorly. In the latter position there is frequently blowing breathing and bronchophony due to compression of the lung by the distended pericardial sac, which resembles closely the signs of a true pneumococcic infiltration; however, the signs of a greatly enlarged pericardial sac anteriorly, without the symptoms of pneumonia, will usually make the diagnosis certain.

There is often dullness in the chest posteriorly; the dullness may be so great that the effusion may be thought to be in the pleural cavities.

Unless the pressure within the cardiac sac is great, however, the radial pulse remains remarkably full.

The physical signs when the condition is due to a transudate are much the same.

Conditions to be Differentiated from Pericardial Effusion

The condition is to be differentiated from:

Cardiac dilatation
Cardiac hypertrophy
Pleural effusion
Chronic adhesive pericarditis.

CARDIAC DILATATION.

In cardiac dilatation there is enlargement of cardiac dullness. Here the dullness is as large or larger at the base than at the apex, in contradistinction to pyriform shape in effusion. Diffuse cardiac impulse, feeble and irregular pulse, enlargement of the liver, dyspnea, palpitation of the heart, and edema are its principal signs. The cardiac impulse is as great, or greater, in size than it is in an effusion, but the impulse of the heart may be found extending over almost the entire cardiac area. Murmurs are heard in contradistinction to the muffled and distinct heart sounds of effusion. The symptoms—edema, palpitation and dyspnea—are much more marked as a rule than in effusion.

CARDIAC HYPERTROPHY.

In this condition there is increased dullness and loud first sound with forcible heart beat, as distinguished from the indistinct feeble heart sounds of effusion.

Perhaps the most difficult condition to differentiate is that of a *dilated heart,* without a murmur at any one of the orifices.

In this the differences are largely those of history and physical signs. In pericardial effusion due to acute pericarditis there is the history of fever, pain, friction sound and an acute infection; these are entirely wanting in the dilatation of the heart due to primary myocardial change. Here there is the history of dyspnea on exertion, slight edema, and frequently of pain in the precordium due to exertion. The physical signs are increased cardiac dullness, feeble and often irregular heart sounds with the muscular element but slightly in evidence. There is a feeble pulse, often curiously different from the rather full pulse present in pericardial effusion. The apex beat when felt is far to the left of its normal position, while in effusion it is usually lifted above the normal position.

PLEURAL EFFUSION.

An error is likely to occur only in a left-sided effusion.

In pleural effusion the heart is usually pushed over to the right; this is not the fact in pericardial effusion. The history usually differs in these conditions. An x-ray picture, and particularly a fluoroscopic examination, will furnish much help.

CHRONIC ADHESIVE PERICARDITIS.

This condition is difficult of diagnosis because of the dearth of physical signs. As a rule the condition is known by the signs of cardiac decompensation which come on as the end picture of the disease.

10. Acute Endocarditis

Origin.—This is the result of some general infection—acute rheumatism, tonsillitis and gonorrhea being the most common causative factors, or it may arise as the end result of some chronic condition.

It is of some importance from a therapeutic standpoint to discover the character of the infecting agent and the source of the local infection if such exists.

Symptoms—Diagnosis.—The symptoms of this condition vary in degree; they may be very slight or very severe. It has always seemed to me that it is impossible to surely differentiate between the mild and severe cases by the symptoms present at the time of examination. What seems to be a mild case in the beginning may become truly malignant and cases severe in the beginning may end without great injury to the valves.

Unless the heart is so affected that it becomes rapidly decompensated, valvular involvement may be quite extreme and yet the case have only the symptoms of the original infection—rheumatism, tonsillitis, scarlet fever, etc. Such being the case routine, daily examination of the heart of every individual who has any infection is the only possible method by which every case of endocarditis can be diagnosed in its early stages. For instance, a patient may have slight fever with joint pains, and no evidence of cardiac disturbance may be present in the symptoms of the case; examination, however, will at once show a murmur and possibly an enlargement together with irregularity of the action of the heart. This diagnosis is of the utmost importance in the therapeutics of the case. Embolism in the spleen or kidney, or indeed in any portion of the arterial tree may occur, giving rise to symptoms referable to disturbance of the organ affected, or, if the embolism is small, there may be no symptoms whatever. Absolute quiet for a prolonged time is the most important therapeutic measure.

The severe cases and the so-called malignant cases are marked by prolonged fever, often of intermittent type, with anemia, petechia, and later with severe cardiac symptoms, which latter unfortunately are often the first symptoms to attract attention to the cardiac involvement. These symptoms of fever so often dominate the case, that the observer frequently considers the case one of tuberculosis or typhoid fever until severe cardiac symptoms develop or death supervenes.

These cardiac symptoms are oppression under the sternum, seldom true pain in the cardiac region, irregular pulse at times, usually rapid. An endocardial murmur dependent upon the valve affected for its time and rhythm is almost always present. It must be remembered, however, that a murmur can be present without an actual inflammation of the endocardium; it may be a pericardial murmur when it is always to-and-fro, heard over almost the entire heart, and always without reference to the valve affected by the endocarditis. It may be a murmur due to simple dilatation, not the result of any endocardial complication, but of myocardial weakness. In the severe cases emboli are more common than in the mild; these emboli are apt to become suppurative, converting the case into one of veritable pyemia.

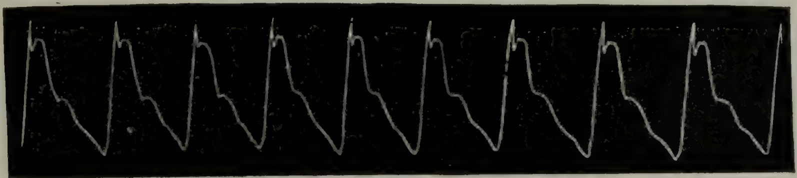


Fig. 85.—Pulse of Slight Aortic Regurgitation with Good Heart Muscle. (After Mackenzie.)

Conditions to be Differentiated from Acute Endocarditis

The severe cases may be mistaken for:

Malarial fever
Ordinary septicemia
Pernicious anemia
Typhoid fever
Tuberculosis.

Malarial Fever.

Malarial fever of the tertian type could scarcely be mistaken for endocarditis except by the very careless, but in irregularity the intermittent fever of estivo-autumnal fever does resemble the fever of endocarditis at certain times. However, in malaria the specific organism is always present, there is no leukocytosis, no heart murmurs or other evidences of cardiac involvement, while in endocarditis leukocytosis is present, there are no malaria plasmodia, and the cardiac symptoms are evident. The spleen may be enlarged in both diseases.

Ordinary Septicemia.

Ordinary septicemia may have exactly the same symptoms and etiology as endocarditis with the exception of the cardiac symptoms—they are absent. The differentiation then must depend upon the absence of cardiac murmurs, and of cardiac dilatation and the presence of a focal lesion to account for the sepsis.

Pernicious Anemia.

Pernicious anemia resembles endocarditis only in its severe anemia. In pernicious anemia there are the characteristic signs in the blood of many nucleated red cells, poikilocytes, normoblasts and megaloblasts; there is no fever, no leukocytosis, but usually a leukopenia with a relative increase in the lymphocytes is observed. There are cardiac murmurs in anemia, but these are soft and *not rough* in character, and the dilatation which is often extreme in severe cases of endocarditis is much more moderate in pernicious anemia.

Typhoid Fever.

Typhoid fever might be mistaken for endocarditis, though usually the mistake is made of mistaking endocarditis for typhoid fever. In typhoid there is leukopenia, in endocarditis leukocytosis. If the case is one of typhoid, a blood culture will show typhoid bacilli, and not other organisms; a Widal reaction will be present. Cardiac involvement rarely occurs in typhoid fever.

Tuberculosis.

Tuberculosis has been mistaken for endocarditis, and the reverse diagnosis has been made. Only irregular and intermittent fever and a pulmonary infarct in the course of endocarditis will account for the mistake.

In tuberculosis there is always a local lesion outside of the heart. The lung lesions of endocarditis come on suddenly as the result of emboli, those of tuberculosis gradually. Tubercle bacilli occur in tuberculosis and not in endocarditis.

11. Aortic Regurgitation

Symptoms.—In aortic regurgitation the symptoms are headache, vertigo, tendency to syncope, cardiac palpitation, pallor of the face, head nodding, alternating flushing and paling of a portion of the surface of the body, and edema. Sometimes there are no symptoms until decompensation sets in. Anemia occurs and pains of true angina pectoris are not uncommon.

Physical Signs.—The physical signs are, principally, a diastolic murmur lasting through the second cycle of the heart. It is heard best at the left third intercostal space near the sternum and conducted down the left edge of the sternum; it is often best heard in the region of the xyphoid. The lesion is often accompanied by the so-called Flint murmur—a presystolic murmur, the result probably of the stream of regurgitated blood impinging on the mitral leaflet. It is only present when there is an aortic regurgitation. This Flint murmur can be distinguished from the presystolic murmur of mitral stenosis only by the symptoms and signs which belong to the latter condition. Aortic regurgitation may also be accompanied by a true mitral systolic murmur, the result of relative insufficiency. There is often great hypertrophy of the left ventricle with diffuse cardiac impulse together with rapid receding of the radial pulse, the latter more marked when the hand is raised above the head. A capillary pulse can be seen under the finger nails and upon the mucous membranes of the lips when they are lightly compressed by a glass slide, or it may be seen as an alternating flushing and paling when the skin of the forehead is vigorously rubbed by a towel or rather rough cloth. The sound of the heart can frequently be heard over an artery uncompressed. A sound resembling a pistol shot is often heard over the femoral artery

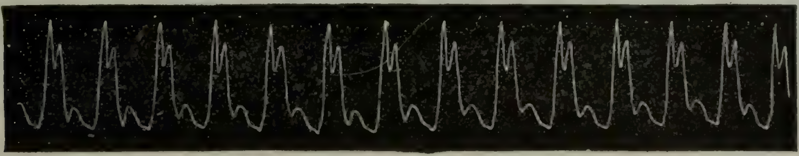


Fig. 86.—Pulse of Aortic Regurgitation with Great Cardiac Failure. (After Mackenzie.)

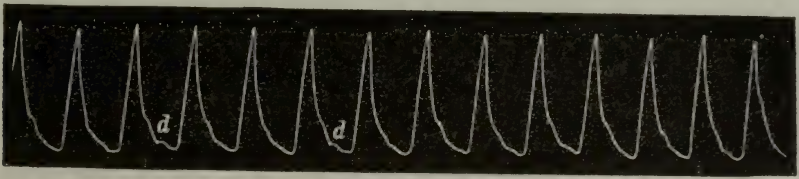


Fig. 87.—Pulse of Extreme Aortic Regurgitation with Great Cardiac Failure. (After Mackenzie.)

near Poupart's ligament. It is in aortic regurgitation that the largest hearts frequently occur. Patients usually have little edema until a secondary failure of the mitral orifice occurs; they then take on all of the symptoms of dyspnea, liver enlargement and general anasarca common to mitral and tricuspid regurgitation. The pulse tracing is characteristic.

Conditions to be Differentiated from Aortic Regurgitation

It might be mistaken for:

Aneurism of the arch of the aorta
Mitral stenosis
Double aortic disease.

ANEURISM.

In aneurism there is little hypertrophy of the heart, no capillary pulse, no pulsation under the sternum; a diastolic shock and often a thrill and a tracheal tug are present. When there is a double murmur, the diastolic element is not long drawn or conducted in the manner of an aortic regurgitation. Lastly, an x-ray will show an abnormal shadow in the line of the aorta.

MITRAL STENOSIS.

This condition might be confused with mitral stenosis because of the diastolic murmur occurring in mitral stenosis when the heart is the subject of auricular fibrillation; also when a Flint murmur is present in aortic regurgitation.

DOUBLE AORTIC DISEASE.

Aortic regurgitation and aortic stenosis, or a roughening at the orifice in the beginning of the aorta, very frequently occur in the same heart. Here there is a marked double murmur. The systolic murmur is conducted into the vessels of the neck. If the systolic murmur is due to a true stenosis of the orifice there is an hypertrophy of the heart, greater than in a simple regurgitation, and the systolic murmur is usually louder and rougher than when due to a simple roughened orifice or an aortitis; in addition, there is very apt to be a systolic thrill over the aortic cartilage.

12. Aortic Stenosis

In aortic stenosis there is greatly enlarged *left* heart, a small radial pulse, a rough systolic murmur conducted into the vessels of the neck best heard at the right third intercostal space. Frequently there is a palpable thrill systolic in time accompanying the murmur and there may be a very distinct subternal pulsation. The pulse tracing is characteristic.

Conditions to be Differentiated from Aortic Stenosis

Simple aortitis, or roughening of the aortic ring
Aortic aneurism
Functional basic murmurs.

Simple Aortitis.

Simple aortitis, hemic murmurs at the base, and roughening of the aortic orifice are often mistaken for aortic stenosis. They can be distinguished from that condition by the absence of hypertrophy of the left ventricle and also by the absence of a feeble radial pulse, both of which occur in aortic stenosis and both of which are absent in simple roughening. The simple presence of a systolic murmur, best heard over the aortic orifice, is *not sufficient* evidence upon which to base a diagnosis of aortic stenosis.

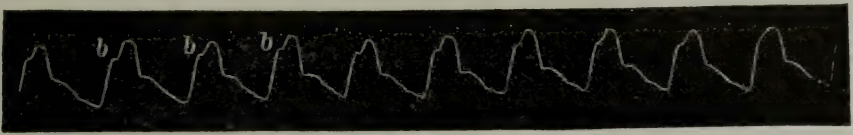


Fig. 88.—Anacrotic Pulse. From a Case of Aortic Stenosis. (After Mackenzie.)

Aortic Aneurism.

Aortic aneurism may be the diagnosis in certain cases of aortic stenosis. This mistake can occur only when there is dullness under the first piece of the sternum together with the presence of a thrill and pulsation in certain cases of aortic stenosis. Aneurism, however, is differentiated by means of the bell-like character of the second sound, a diastolic shock and lack of hypertrophy of the left ventricle and above all by a fluoroscopic examination and the picture on a plate. Here the aorta will be shown to be dilated separately from the heart in aneurism, and the heart alone enlarged in stenosis.

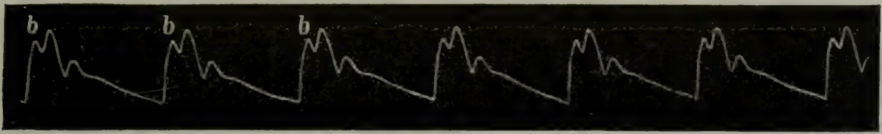


Fig. 89.—Pulsus bisferiens. From a Case of Aortic Stenosis. (After Mackenzie.)

Functional Basic Murmurs.

Hemic murmurs are not loud; there is evident anemia; there is no thrill and no hypertrophy of the heart. There however may be dilatation of the heart due to weakness of the heart muscle.

13. Mitral Regurgitation

Symptoms and Physical Signs.—Mitral regurgitation due to endocarditis is characterized by enlargement of the heart both to the right and left with a systolic murmur best heard at the apex and conducted toward the axilla and around to the shoulder. Symptoms occur when decompensation begins, the symptoms being due to coincident tricuspid regurgitation; they are cyanosis, palpitation and edema.

Differentiation.—It may be confused with REGURGITATION which occurs as the result of a relative dilatation which is not caused by disease of the mitral valve. It may be confused with an extension of the heart dullness to the left, the result of the heart either being pushed over by a collection of fluid or a new growth in the right chest. In regurgitation which is due to relative dilatation and not due to organic change in the valve opening, there may be the history of cardiac difficulty before the appearance of the murmur, or there may be the history of a perfectly sound heart before the appearance of the cardiac failure. In the latter instance the failure must be the result of some sudden strain put upon the heart.

In relative insufficiency by rest, and possibly the use of the cardiac stimulants such as digitalis, the murmur will disappear with the return toward normal of the strength of the heart. If the murmur is due to change in the mitral leaflets, the murmur will increase in intensity with the return of strength of the heart muscle. This observed fact will place the diagnosis between a relative regurgitation and one due purely to an organic lesion.

When the heart is pushed to the left the presence of dullness, lack of breath sounds and voice sounds over the right chest should lead to the diagnosis of an abnormal position of the heart. The murmur may be exactly simulated by a systolic regurgitant murmur due to insufficiency of the tricuspid valve. This may be particularly the case when the tricuspid regurgitation has its primary cause in mitral stenosis. The differentiation must depend upon either the presence of a mitral stenotic murmur or a sharp first sound characteristic of mitral stenosis, and above all upon the fact that a marked pulsation in the jugulars indicates the tricuspid origin of the murmur.

14. Mitral Stenosis

Symptoms and Physical Signs.—This valvular defect has a somewhat characteristic symptom in the early stages of decompensation, i. e., the tendency to severe dyspnea and to unexpected attacks of severe pulmonary edema after some severe or unusual condition, such as after an ordinary delivery of a child, walking in the high wind, etc. It is more common in women.

Its physical signs are hypertrophy of the right ventricle and left auricle, little dilatation of the left ventricle, a very sharp accentuated first sound of the heart, a presystolic murmur often rough and rumbling in character, a sharp snappy character of the first sound of the heart and a presystolic thrill.

Differentiation.—These signs positively mark a mitral stenosis. It might be possible to mistake the accentuated first sound for the second sound of the heart and thus confuse the rhythm, and in consequence, making a diagnosis of MITRAL REGURGITATION. Flint's murmur or the presystolic murmur which sometimes accompanies aortic regurgitation might be mistaken for the murmur of mitral stenosis which it simulates exactly in time and frequently in character. In Flint's murmur, however, there is *always* an aortic regurgitation; there is lacking the hypertrophy of the *right* ventricle, and also the accentuation of the first sound. Usually there is no palpable thrill with the Flint murmur.

The polygraph assists greatly in the diagnosis.

15. Tricuspid Orifice Regurgitation

Etiology.—This condition is much more frequently due to stretching of the tricuspid orifice than it is to an inflammatory condition of the valves. When the latter is the case, when it is due to a valvulitis, it is either congenital or due to endocarditis, which has affected the tricuspid valve, and there are no signs of disease at other orifices of the heart.

Mitral regurgitation and mitral stenosis, or a dilatation of the heart, the result of high arterial tension, is the common cause of the secondary tricuspid regurgitation. Regurgitation of the blood through the tricuspid orifice is evidenced by the pulsation of the veins of the neck, enlarged liver and marked cyanosis. Occasionally the regurgitation is so marked that there is a pulsation in the epigastrium due to the impulse of the heart in this position and either a transmitted pulsation of the liver or an actual pulsation of the liver itself.

There is marked edema of the legs and feet; further, there is a loud systolic murmur over the epigastrium, which is conducted toward the right and not the left.

Differentiation.—Tricuspid regurgitation may be mistaken for MITRAL REGURGITATION in cases of long standing, in which the real lesion is that of mitral stenosis. Here the anterior portion of the heart which gives rise to the heart dullness is really composed of the right ventricle and the right auricle of the heart (Fig. 90). The loud systolic murmur closely resembles a mitral regurgitation and often entirely obscures the mitral stenotic murmur, but the enlarged liver (sometimes pulsating), the tendency to pulmonary edema, and the systolic pulsation in the vessels of the neck will enable one to make the diagnosis of tricuspid regurgitation, while the marked enlargement of the heart upward will help to diagnose mitral stenosis. Particularly valuable as a differential sign is the sharp accentuated first sound maintained in cases of mitral stenosis, even though the presystolic murmur be lost.

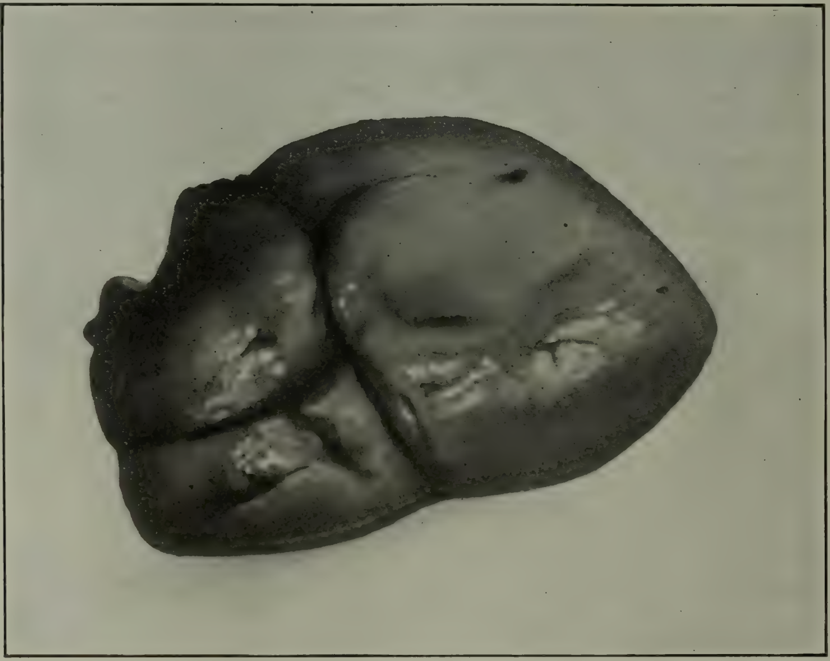


Fig. 90.—Heart in Extreme Mitral Stenosis. The Right Auricle and Right Ventricle Form the Entire Anterior Surface of the Heart. The Heart Lay Almost Transversely in the Chest. (Original Observation.)

Mitral regurgitation is frequently accompanied by tricuspid regurgitation as the result of dilatation of the right ventricle, and can be diagnosed by the symptoms and signs noted above.

Mitral regurgitation itself may be differentiated by a systolic murmur conducted to the axilla and scapula, enlargement of the heart to the left, and the absence of the cyanosis, edema, right heart enlargement and enlargement of the liver.

16. Tricuspid Stenosis

Tricuspid stenosis is extremely rare and a correct diagnosis made during life is still more so. It is seldom the result of rheumatism.

Symptoms and Physical Signs.—The murmur is presystolic in time, and is best heard in the center of the sternum; there is dilatation of the right auricle of the heart and accentuation of the first sound of the heart.

The symptoms are cyanosis, dyspnea, great enlargement of the liver due to passive congestion. The area of heart dullness is far to the right of the sternum and is not upward; the increase of dullness to the right is due to dilatation of the auricle; there is a presystolic pulsation in the jugular veins.

Differentiation.—Mitral stenosis, and mitral stenosis in the late stages may be mistaken for the condition.

Mitral stenosis is the result of a rheumatic infection. There is great enlargement upward of the heart dullness, owing to the dilatation of the left auricle. The murmur is near the apex or is conducted toward the axilla. There is often a very loud systolic murmur due to mitral regurgitation owing to the relative dilatation of the right side of the heart.

Mitral regurgitation after the right side of the heart is much dilated might possibly be mistaken for tricuspid stenosis because of the increase of heart dullness to the right and general venous stasis, but there is no presystolic murmur and the regurgitant murmur is conducted far over toward the axilla.

17. Pulmonary Valve Disease

Pulmonary stenosis is extremely rare and is almost without exception a *congenital lesion.* When a loud murmur at the pulmonary orifice, systolic in time, is accompanied by hypertrophy of the right ventricle, with signs of congenital heart disease—cyanosis, clubbed fingers and stunted growth of the individual, a diagnosis can reasonably be made of stenosis of the pulmonary orifice. The disease being extremely rare, other conditions are constantly mistaken for it. The occurrence of the disease is still more unusual, without other signs of congenital lesion than the murmur.

Conditions to be Differentiated from Pulmonary Valve Disease

It can be mistaken for:

Pulmonary murmur due to anemia
Pulmonary murmur due to adhesions between the orifice and the pleura
Cardiorespiratory murmur
Pulmonary murmur present as the result of a rapidly acting heart in fevers
Aortic stenosis.

Murmurs Due to Various Causes.

A diagnosis of pulmonary stenosis should not be made unless the common sources of pulmonary murmurs are positively excluded.

All of the latter conditions lack hypertrophy of the right ventricle; they all lack any of the signs above stated of congenital disease. *Anemic murmurs* are accompanied by a distinct anemic condition of the blood, loss of hemoglobin, of red cells, or both. These conditions are not present in pulmonary stenosis.

A cardiorespiratory murmur can be made to entirely disappear or at least be distinctly modified by deep inspiration or full expiration. This does not affect the murmur of pulmonary stenosis.

The murmur in a rapid heart with fever is so manifestly connected with an acute diseased condition, that it can scarcely be confused.

The main points to be remembered, therefore, are the extreme rarity of a true pulmonary stenosis, with the necessity of hypertrophy, cyanosis, etc., as a part of the physical signs and symptoms of the condition, and the extremely common occurrence of pulmonary murmurs due to other conditions.

AORTIC STENOSIS.

In aortic stenosis there is a small radial pulse as compared with the greatly hypertrophied heart. There is increase of dullness to the left of the sternum; a pulmonary second sound can be heard.

18. Pulmonary Insufficiency

Occurrence.—Pulmonary insufficiency, like pulmonary stenosis, is an extremely rare condition. It occurs as a *congenital* condition, and occasionally as the result of malignant endocarditis.

Symptoms.—Its symptoms are those of a diastolic murmur in the pulmonary area not accompanied by capillary pulse, water hammer pulse, or hypertrophy of the *left* ventricle.

Differentiation.—When symptoms appear there is failure of the right ventricle exactly comparable to failure of that cavity to tricuspid insufficiency. It is distinguished from this condition by the absence of any disease of the left heart, high blood pressure or any condition giving rise to it.

This condition can also be confused with AORTIC REGURGITATION. Here, as suggested above, there is hypertrophy of the left ventricle, there is a capillary pulse and a water hammer pulse which are not present in pulmonary regurgitation.

19. Congenital Heart Disease

As the name implies, this condition originates previous to birth, being caused by defects in the development of the heart or an actual prenatal endocarditis. The defect can sometimes be discovered before birth by the detection of a murmur in the fetal heart sounds.

In a volume of this kind it is scarcely wise to attempt the differentiation of the multitude of lesions which may occur congenitally, but it is of importance to recognize the distinguishing signs between a *heart lesion which is acquired and one which is congenital.*

Even though a case be seen late in life the *marked cyanosis,* chronic in character, is quite characteristic of congenital disease. The cyanosis of acquired disease is as a rule accompanied by signs of marked decompensation in addition to the cyanosis, palpitation, edema and dyspnea, while the cyanosis of congenital disease usually—but by no means always—is free from other signs of decompensation. This, as would be expected from the causation of the cyanosis, is not a real sign of decompensation. Cyanosis in children not accompanied with cardiac enlargement, is usually the result of some congenital defect.

There is often increase in the number of red cells in the peripheral circulation; there is a murmur which frequently increases as time elapses; it is usually systolic in time; frequently the area of cardiac dullness is not increased, though it may slowly enlarge with the age of the child. The congenital murmurs as a rule are heard best at the base; apical murmurs are rarely congenital. There is often clubbing of the fingers.

Differentiation.—The CYANOSIS DUE TO THE USE OF COAL TAR PREPARATIONS might be mistaken for this condition, as might possibly ARGYRIA, but the examination of the heart will at once show an absence of heart lesions, there being no dilatation, no murmur and no palpitation.

CARDIAC DISEASE DUE TO ENDOCARDITIS AFTER BIRTH has usually the history of some infectious disease. There is no cyanosis unless there is much decompensation; there is no increase in the red cells of the blood.

20. Angina pectoris

Angina pectoris is characterized by painful conditions in the region of the heart or in the epigastrium. This pain is precipitated or increased by exertion and excitement; it is often radiated to one or both arms and may be deflected to other positions of the body. It is of such a character that any exertion the patient may be engaged in at the time must at once be stopped, the sufferer being convinced that a continuation of the act would cause death. Occasionally in the severe paroxysm there is blanching or flushing of the face, accompanied by feeling of compression about the heart and a sense of dissolution.

It usually attacks persons past middle life; there is sclerosis of the arteries and frequently, though not always, there is high blood pressure.

Conditions to be Differentiated from Angina pectoris

The condition is to be differentiated from the pain of
Thoracic aneurism
Intercostal neuralgia
Indigestion—Gastric ulcer—Gall-stones—Appendicitis
A nervous condition (pseudo-angina).

Thoracic Aneurism.

Thoracic aneurism is marked by a dull area in the line of the thoracic aorta, usually a pulsation, and a murmur double in character; often the pulsation can be seen or felt in the suprasternal notch. There is a diastolic shock and often a tracheal tug. The pain which is common in thoracic aneurism, too, is more apt to come on at night, and is extremely likely to be independent of exercise; it is frequently continuous, and often is conducted in the line of the nerve trunks upon which pressure is being made.

While the heart may be greatly disturbed in aneurism, there is no likelihood that any signs which at all approach those of angina pectoris in severity, will appear except pain.

Intercostal Neuralgia.

Intercostal neuralgia is entirely wanting in physical signs; it occurs at all ages; there is pain and tenderness along the line of the intercostal nerve. There is no relation to exertion, though quick movement may increase the pain. There is no cardiac disturbance.

Indigestion—Gastric Ulcer—Gall-stones—Appendicitis.

Cases of angina pectoris occur which have as their cardinal symptoms pain in the epigastrium after eating and on exertion, the exertion having to be stopped immediately. Relief of this pain accompanied by belching of large amounts of gas are frequently considered both by the laity and by physicians as *indigestion.* It will be observed, however, that such attacks of pain have to do with *exertion* and *not with food.* It may be that the attack may come on more severely upon exertion which comes on after eating, but surely it is the exertion and not the food which causes the pain. In cases of angina, the heart is rarely exactly normal. There is also frequent change in the tracings of the polygraph and electrocardiograph.

Such cases, I repeat, are due to real cardiac disease and while they are looked upon often as simple indigestion, they are really cases of angina, and may eventuate in cardiac decompensation or in sudden death. In indigestion the heart is normal, the pain is directly dependent upon the food. If the indigestion is due to gastric ulcer, gall-bladder disease, or to

Angina Pectoris—Differential Chart

	Condition of Heart	Character of Pain	Blood Pressure	Digestive Symptoms	Percussion Signs of Heart	Auscultatory Signs of Heart	Palpation Over Heart	Position of Pain
Anginal pectoris...........	Weak, often irregular	Severe; worse on exertion, ceases on rest	Often high	Belching after attack	Often increase of dullness	Frequently aortic lesions	No thrill unless due to aortic lesion	Into arms
Thoracic Aneurism..........	Normal	Boring; worse at night; often conducted to both arms	No change	None	Heart dislocated; dullness along aorta	Heart sounds normal; pulsation murmur; thrill; shock, over mass	Heart displaced down and outward	Along nerve trunks
Intercostal Neuraliga......	Normal	Short, not affected by exertion	No change	None	None	Normal	None	Along nerve or nerves affected
Gall-bladder Disease, Gastric Ulcer, Appendicitis, Indigestion...............	Normal	Relation to food	No change	Always present	Normal	Normal	None	Usually over epigastrium

appendicitis, there will be the characteristic symptoms of these conditions. It should be a rule of diagnosis that *indigestion lasting several months, which has been treated rationally, has some organic basis for its existence.* One of the lesions is cardiac disease giving rise to angina pectoris.

NERVOUS EXCITABILITY.

Individuals who are hysterical and who are introspective very frequently are subject to paroxysms of pain which to their excited imaginations have to do with some disease of their heart; they frequently complain bitterly of this pain radiating to the arms. In such cases the heart's action may be rapid in the attack, but it is otherwise normal; the blood pressure is normal. There is great excitement. The attack lasts much longer than true angina. The patient is restless and moves about, instead of being forced, by the necessities of the case, to remain perfectly still. The polygraph tracings are normal.

21. Hypertrophy of the Heart

Cause.—The condition is the result of overwork, of chronic valvular disease, or of high blood pressure due either to arteriosclerosis or some toxic condition.

Physical Signs and Symptoms.—This condition is shown by enlarged cardiac area and by forcible cardiac impulse. The sounds are loud and may or may not be accompanied by a murmur, depending upon whether the hypertrophy is due to a valve lesion or not.

There is an actual thickening of the walls of the heart with enlargement of the cavities in certain cases, and normal-sized cavities in other cases.

Conditions to be Differentiated from Hypertrophy of the Heart

It is confused with:

Dilatation of the heart
Pericardial effusion.

DILATATION OF THE HEART.

In dilatation there is enlarged cardiac area, but the sounds are feeble and often irregular. They may be accompanied by a murmur, the result of the dilatation, or it may be the result of some valvular disease. There is lack of compensation as shown by irregular action of the heart, dyspnea, edema and cyanosis.

PERICARDIAL EFFUSION.

There is enlarged cardiac area also in pericardial effusion, but the heart sounds are distant and feeble; the apex beat is often felt above the normal position; there is cardiac distress and sometimes signs of decompensation.

22. Cardiac Dilatation

Etiology.—This condition is indicated by increase of the area of cardiac dullness with diffuse cardiac impulse. This results from all forms of chronic endocarditis, from myocarditis and continued high blood pressure. It is not uncommon in nephritis; it is often the result of emphysema of the lungs and of asthma. Usually it is a manifestation of lack of tone in the cardiac muscle or of actual degeneration of the muscle.

It occurs as an acute condition, the result of cardiac overstrain (usually in a deranged heart), or it may occur in athletes in the beginning of training or in those not properly trained undergoing sudden or severe exercise. Here the heart is usually dilated. Getting the "second wind" is the recovery from a moderate dilatation during exercise.

When the heart muscle itself is severely damaged there is dilatation of the cavities. This occurs in severe fevers, anemia, and in myocarditis. It may be acute under these conditions.

Symptoms.—The symptoms are those of loss of reserve power of the heart—a tendency to dyspnea or palpitation on exertion.

Differentiation.—It is to be distinguished from hypertrophy and pericardial effusion in the manner pointed out above.

23. Fatty Heart

Fatty heart, the pathological change being fatty infiltration or fibroid change, is shown by arhythmia, dyspnea and palpitation on exertion. It may be diagnosed when the symptoms are present with little or no dilatation, when there is an extremely feeble first sound in elderly persons, and when the cardiac action is very irregular.

Differentiation.—From FIBROID DEGENERATION OF THE CARDIAC MUSCLE it cannot be differentiated clearly; indeed the two lesions are usually associated. Fatty infiltration is suggested by the fact that there are fatty deposits in different parts of the organism. The individual is fat.

It resembles:

Cardiac decompensation from chronic endocarditis
Myocarditis due to other causes.

From chronic endocarditis it can be differentiated by the absence of dilatation and by the entire absence of murmurs in the early stages. Murmurs may disappear in chronic endocarditis in the presence of great dilatation but they recur as the heart regains its strength; the dilatation following endocarditis is much greater in extent than that of fatty heart.

24. Cardiac Decompensation

Etiology.—This condition is the result of a disproportion between the work required of the heart and the power of the latter to perform such work. It is at times the result of overstrain from exercise; it may be caused by high blood pressure; it may result from endocardial changes and changes in the pericardium which bring about degeneration of the cardiac muscle; or it may be due to myocardial changes the result of primary degeneration of the muscle or myocarditis, due to disease.

General Considerations.—The normal heart has a certain amount of reserve power. This, for instance, is shown by the fact that the heart in a perfectly normal individual beats about 72 while the person is at rest. When a person runs the heart beat is increased and the individual becomes somewhat dyspneic; but on again coming to rest the heart beats as before the exercise and the individual is perfectly comfortable. The reserve power resident in his heart has allowed that organ to respond without damage to health. If, however, the patient has a heart with little or no reserve power and attempts to run, he is entirely unable to do so with comfort, but if he should persist in the violent exercise he will become extremely dyspneic, perhaps cyanosed, and it will take a long period of time before his heart is again quiet—indeed his life may be endangered by such exertion.

The symptoms of this loss of heart reserve power are indicated by breathlessness, faintness, cardiac palpitation, edema of the extremities, enlargement of the liver; it sometimes results in slow or sudden death. There are marked symptoms of indigestion due to stasis in the vessels of the alimentary canal, vertigo and at times syncope, together with pain in the region of the heart, sometimes anginal in character. Sometimes there is a gallop rhythm; albumin and tube casts often appear in the urine. There is usually enlargement of the cardiac area, with diffuse cardiac impulse. However, in angina pectoris, which is certainly one form of cardiac incompetency, there may be no change in the heart size, though usually there is dilatation or hypertrophy, or both.

Conditions to be Differentiated from Cardiac Decompensation

Hysteria
Nephritis.

Hysteria.

Some nervous condition, such as hysteria, can simulate cardiac decompensation. Here the heart sounds are normal; the rhythm is normal though the heart may be rapid; there is no albumin, no digestive disturbance which can be considered as related to the attack. The patient will often recover if left alone.

NEPHRITIS.

These same symptoms of cardiac decompensation are in a certain degree present in severe nephritis. In this condition the cardiac muscle is itself fairly competent, or is secondarily affected and the valves normal. In both cases tube casts and albumin may be present. The edema in nephritis is due to the altered condition of the blood, and the cardiac distress, to the toxemia present. Which of these conditions is present is a serious question to decide, and one upon which the therapeutics of the case depends. As a general statement it may be taken that cardiac disease is the cause of the symptoms when palpitation of the heart, dyspnea, cardiac enlargement and edema are early symptoms, and where examination of the urine at first showed no abnormality. On the other hand, if the edema and urinary derangements are the first symptoms noticed, cardiac enlargement, murmurs, dyspnea, cyanosis and palpitation occurring later, then the condition is much more likely to be primary in the kidney. Albumin and tube casts are present in both. Where the kidney is primarily at fault there is very low phthalein output; there is more albumin and tube casts. The albumin and tube casts do not disappear, though they may grow less when the case ameliorates.

The renal insufficiency, while present in cardiac disease, is neither so severe nor persistent as it is in primary renal condition, and the albumin and tube casts disappear with the improvement of the case.

25. Aneurism

Definition.—An aneurism is a more or less sacculated dilatation of an artery, as distinguished from a uniform dilatation of the vessel.

Cause.—Its cause is a lesion of the intima, frequently syphilitic in origin, which brings about a destruction of that membrane, weakens the vessel wall at the point of lesion which allows a local stretching of the coats, and finally leads to a rupture of one or all of the coats of the vessel.

Occurrence.—It occurs most frequently in individuals who are under prolonged physical strain, soldiers and carpenters being among those oftenest affected. It may take place in any artery of the body, but it is much more common in vessels unprotected by surrounding structures, as in the thorax, the brain and the popliteal space.

The aneurisms of the greatest importance from a diagnostic standpoint are those affecting the arteries of the thorax and the abdomen.

Thoracic Aneurism

Aneurism of the thoracic aorta may affect any portion of that vessel from its beginning at the heart to its exit through the crura of the diaphragm. It may give no subjective symptoms.

The most frequent position is some portion of the arch of the aorta. The ascending and transverse portions are the most commonly affected.

Diagnosis.—Perhaps the most important method of all which helps in a diagnosis of aneurism of the aorta, is the *use of the x-ray.* Constantly, when physical signs are meager, the case difficult of solution, the presence of a shadow on an x-ray plate will clear up the entire case.

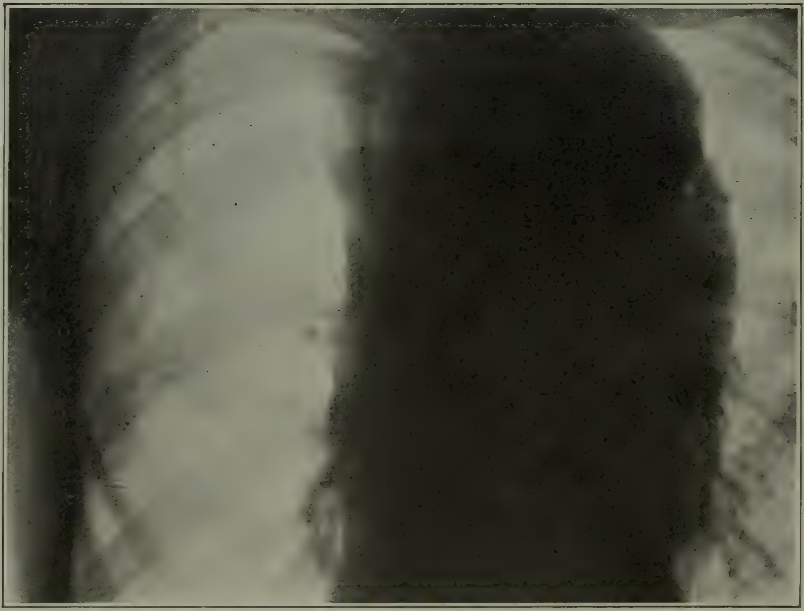


Fig. 91.—Röntgen Ray Photograph of Aneurism of Descending Aorta. (Kindness of Dr. H. K. Pancoast.)

The means of diagnosis are, first, *inspection.* This should be carefully practiced in every suspicious case. If the dilatation is of sufficient size, the sac will impinge upon the chest wall; there will be a fullness, and possibly a pulsation. If the fullness is observed on both sides of the chest it will be greater on one side than on the other (Fig. 92).

All aspects of the chest must be carefully examined. Nothing is more common than for a large aneurism to give absolutely no physical signs in the anterior portion of the chest and yet be quite evident in the back. Such a case is now under my care at the Episcopal Hospital. This case had been variously diagnosed. Persistent pain in the chest led to an x-ray which showed a very large shadow at the site of the transverse and descending arch. Careful examination of the anterior portion of the chest gave absolutely no evidence of an aneurism except a bell-like second sound. Examination of the back, made more carefully than at the first examination, showed fullness at the right interscapular space with a very marked pulsation synchronous with the heart beat.

Palpation is as important as inspection. The hand laid upon the bare chest will frequently distinguish an irregularity in the chest wall or a local pulsation which would not have been noticed by inspection alone. Palpation will frequently discover the pathognomonic diastolic shock. This sign may be described as a short, sharp pulsation synchronous with the diastole of the heart and immediately following the systolic pulsation. It is as though the underlying chest wall were slowly raised by the systolic impulse and then suddenly receded and ended abruptly with a short, sharp impulse against the chest wall. This sign is pathognomonic of aneurism.

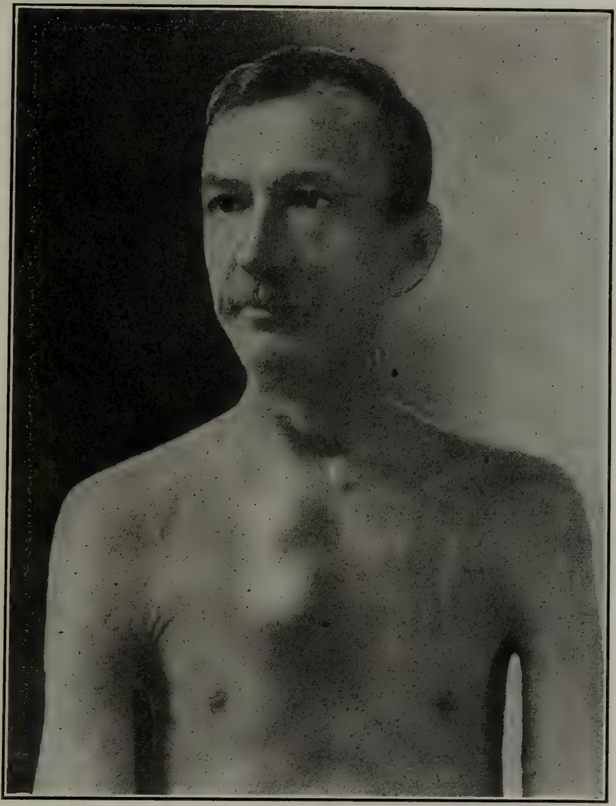


Fig. 92.—Aneurism of the First Part of Arch of Aorta, Protruding Through Chest Wall. (Personal Observation.)

Percussion will show a dullness overlying the aneurismal sac. The percussion must be practiced both lightly and strongly. Light percussion as a rule should be used, but it will frequently not develop a deep-seated aneurism, which may easily be distinguished if the percussion is hard and strong. The area of heart dullness is not very much increased. The heart may be pushed out of place but is little hypertrophied.

Tracheal tug, a systolic pull when the trachea is lifted by the finger, is another important sign. This sign should be developed in the following manner: the terminal phalanx of the index finger is strongly flexed while the finger is extended; the flexed phalanx is then hooked under the cricoid cartilage and the trachea lifted. If the trachea is attached to the aneurismal sac each systolic beat of the heart will pull upon the trachea and give a downward tug.

The habit of grasping the trachea with the thumb and forefinger, and lifting it, is entirely unsatisfactory. A strong pulsation of the carotids may entirely confuse the observer and lead to the diagnosis of a tug when none exists.

Delayed pulse at one or the other wrist, with low blood pressure in the affected side may occur. Dilatation of one or the other pupil may occur because of pressure of the sac of the aneurism upon the sympathetic. Hoarseness, aphonia and brassy cough are signs of pressure due to involvement of the recurrent laryngeal nerve; unilateral sweating may exist as the result of pressure upon the sympathetic nerve. Pain in the course of a thoracic or brachial nerve may be continuous and excruciating—due to pressure upon the trunk of a nerve. Boring pains due to pressure of the aneurismal sac upon some bone, the sternum, or the spinal column or upon the ribs is another important sign.

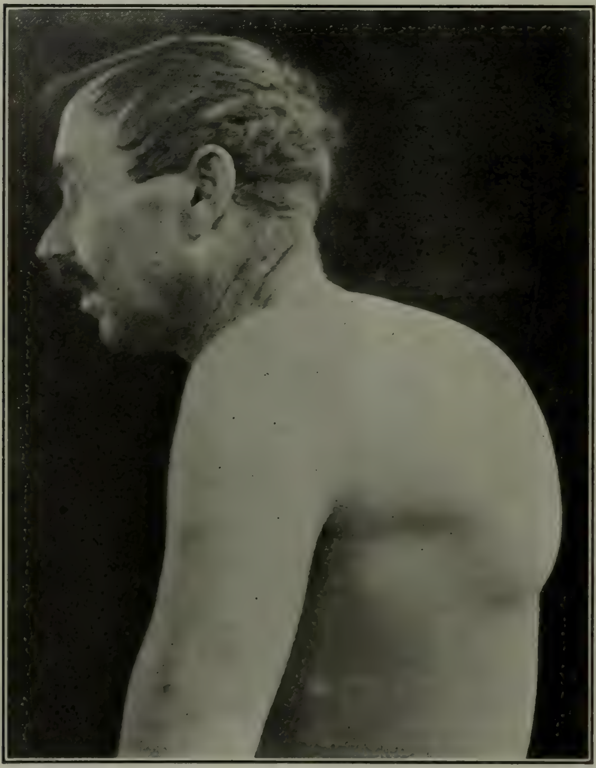


Fig. 93.—Aneurism Pointing in the Back. (Personal Observation.)

A few cases have been reported where the aneurism has pressed upon or ruptured into the superior vena cava. The writer has observed four such cases and reported one. In this instance there is sudden edema and cyanosis of the arms and upper half of the chest, face and neck. If there is really a rupture into the cava there is a high-pitched continuous musical murmur heard over the second and third intercostal space. This set of symptoms, occurring suddenly, is quite diagnostic.

Conditions to be Differentiated from Aneurism of the Thoracic Aorta

Solid tumor of the mediastinum
Abscess or cyst of the mediastinum
Disease of the aortic valves
Simple dilatation of the aorta.

SOLID TUMOR OF THE MEDIASTINUM.

A solid tumor will give a dull note upon percussion. There is usually no pulsation, though a large tumor may give a sensation of pulsation by transmitting the impulse of the aorta upon which it rests. This is rare and the pulsation, when it exists, is not of the same character as that present in an aneurism. In the latter there is the sensation of an expansile body underneath the tissues. This is not the fact of a solid body as when a solid tumor of the mediastinum is present. Tracheal tug is not present in a solid tumor usually, though one can imagine its appearance if there are adhesions between the trachea, the tumor and the arch of the aorta. Diastolic shock is absent in tumor. A pressure cough may be present in a tumor as well as in an aneurism, but there is usually no paralysis of the vocal cords when the pressure is due to tumor.

ABSCESS OR CYST OF THE MEDIASTINUM.

A cyst would closely resemble a solid tumor.

An abscess may give many of the pressure symptoms of an aneurism, but tracheal tug, pulsation and diastolic shock will not be present. A bruit is frequently heard; this bruit is either a to-and-fro murmur or is a simple systolic murmur. In abscess there is fever and leukocytosis.

DISEASE OF THE AORTIC VALVES.

An aneurism is distinguished from aortic regurgitation or stenosis by other physical signs than auscultation. In both regurgitation and stenosis the heart dullness is greatly increased. This, as stated before, is not the case in aneurism of the arch of the aorta. An aneurism of extremely small size may exist at the very beginning of the arch of the aorta and give all the signs of a valvular disease because it will cause interference with the closure of the orifice, yet the valves themselves may be intact.

In aortic regurgitation there is a capillary and a water hammer pulse; neither are present in aneurism. The bell-like second sound, which is so characteristic of aneurism of the arch of the aorta, is replaced either by a murmur or a very impure second sound. The pressure symptoms of aneurism of the arch are not present in regurgitation and stenosis.

SIMPLE DILATATION OF THE AORTA.

Dilatation and tortuosity of the arch of the aorta without a saccular enlargement may simulate a true aneurism. There may be dullness under the sternum with a murmur usually systolic in character. Usually, however, there are other signs; tracheal tug, diastolic shock, irregularity of pulse. An x-ray picture will differentiate this condition from a true saccular aneurism.

Aneurism of the Abdominal Aorta

Aneurism of the abdominal aorta should not be diagnosed without an x-ray picture, a palpable mass, a murmur, and a thrill. The symptoms are pressure and pain. Pressure may be upon the vena cava and cause edema and cyanosis of the lower limbs, as there may be pressure causing erosion of the bones and consequent great pain. There is always a mass which can be distinctly felt which pulsates and which is usually the seat of a thrill and a murmur.

A solid tumor, either a malignant growth or enlarged glands, may give many of the same symptoms. In case the mass is a malignant growth there will be loss of weight and emaciation added to the other symptoms. This procedure may be used to distinguish these two conditions: place the patient upon the elbows and knees to determine whether the pulsation is transmitted or inherent. The pulsation will cease if the mass is not connected with the vessel.

The condition most commonly mistaken for aneurism of the abdominal aorta is an abnormal pulsation of that vessel. Frequently in aortic regurgitation, in extreme anemia, and in neurasthenia, very forcible pulsation of the abdominal portion of the vessel occurs; however, there is never a distinct mass. Careful palpation will show the vessel pulsating but not dilated, extending the normal length through the abdomen. An x-ray will show no shadow in the plate. There may be a thrill and a murmur, but these are only functional and not due to any lesion of the vessel itself.

Peripheral Aneurism

A peripheral aneurism is indicated by a pulsating mass in the course of the vessel. There may be a tortuous and dilated vessel which closely resembles an aneurismal dilatation, but careful examination will indicate difference between the two conditions, and an x-ray will give the final help. A solid tumor, a cyst or an abscess over the line of the vessel may pulsate, but the pulsation will be transmitted; it will not be expansile.

26. Arteriovenous Aneurism

Etiology.—This anomaly is caused by the entrance of the arterial flow directly into the venous flow, and is frequently the result of a wound which connects both the veins and the artery.

There is a pulsating mass which is frequently irregular in shape, and is apt to be tortuous. Over this there is a more or less continuous sound which increases with the systolic action of the heart.

Differentiation.—It may be confused with an ordinary VARICOSE VEIN, but the latter does not pulsate and there is no murmur. A peculiar form of this arteriovenous aneurism occurs at times AS THE RESULT OF AN ANEURISM OF THE ARCH OF THE AORTA INTO THE SUPERIOR VENA CAVA. This condition necessarily follows an aneurism of the arch of the aorta or one of its branches.

The aortic aneurism may be so small that it cannot be recognized, nor does it give any symptoms. Suddenly, however, the patient becomes cyanosed in the face, neck, arms and upper half of the chest.

Over the sternum there is usually a high-pitched to-and-fro singing murmur. The curious appearance of the cyanosed swollen half of the body is most striking, and once seen will never be forgotten. It can be mistaken only for some pressure on the superior vena cava without rupture into the vessel. This may be in the form of a mediastinitis. The author has seen such a case which at autopsy showed a firm, fibrous union under the sternum, due to tuberculosis starting in the peribronchial glands.

In this condition there are none of the signs of aneurism, and the high-pitched singing murmur is conspicuous by its absence.

If a tumor pressing on the veins were to bring about these same symptoms, the murmur would be absent.

27. Arteriosclerosis

This is a general disease affecting the arteries of the body. At times it affects all of the arteries, at other times only the arteries in local areas.

Characteristic Symptoms.—It is characterized by thickening of all coats, and in the larger by gelatinous swelling, necrosis, fatty degeneration and calcification—the process to which the name atheroma has been given (Osler).

The superficial arteries are quite palpable.

Causes.—The causes of arteriosclerosis are well known. They are grouped by Osler under the headings: Wear and Tear of Life, Intoxications, Acute Infections and Circumstances which Keep the Blood Pressure

High. For most entertaining and instructive reading, the student is referred to Osler's article on arteriosclerosis in "Modern Medicine."

The day laborer develops early a condition of the arteries which results in thickening of their walls, tortuous vessels, calcerous patches. He, however, is enabled to continue at his work without much extreme danger; he may or may not have high blood pressure. The modern business man, the hard-working physician, the politician, subjected to mental strain in his efforts to keep up with the advance in his especial calling, develops the same condition of his arteries, and in addition a high arterial pressure, the latter being maintained until some serious break in the circulation appears.

Acute infections, especially syphilis, are accompanied and followed by severe arteriosclerosis with high blood pressure. Other infections, particularly typhoid fever, are also followed by arteriosclerosis and high blood pressure.

Intoxicants, lead, tobacco, alcohol, and the toxins in kindred diseases play a large part.

High blood pressure caused by these toxic substances mentioned above, hypersecretion of the adrenals, the high pressure so characteristic of interstitial nephritis, unquestionably end by causing arteriosclerosis.

Arteriosclerotic individuals do not necessarily have high arterial tension. High arterial tension must be looked upon simply as a symptom. It may be due to diseases of the kidney, a simple temporary condition due to nervous strain; it may be due to overeating, overworking, and overdrinking. It may be the result of a cerebral neoplasm. It may be compensatory, the result of broken cardiac decompensation, and therefore while arteriosclerosis is a rather constant accompaniment of prolonged high pressure one must search in the history of the case for facts and causes, one must make a careful physical examination in order to interpret its meaning.

Symptoms.—The symptoms referable to general arteriosclerosis are manifested by a loss of physical and intellectual vigor in the individual, slight pallor, and finally breathlessness, slight edema, vertigo and irritability manifest themselves—being instances of the failure of special organs.

The *important time to recognize the beginning of arteriosclerosis is early*, when the individual seems to be in perfect health. His arteries are thickened, his blood pressure raised; these are signs of warning to lessen the urgency of work.

The symptoms of arteriosclerosis may be almost nil until some special organ is so affected that its function is interfered with.

A patient may present himself with symptoms referable to almost any organ. Careful, general physical examination, including the estimation of blood pressure and urine examination, is necessary before one can come to a conclusion as to how much general arterial degeneration exists.

Nervous System.—When the vessels of the nervous system are affected there may be vertigo, headache, fleeting palsies. Hemiplegia, aphasia and paraplegia have all been observed; they are transient and soon disappear, not leaving any serious loss of function as do these same symptoms when due to hemorrhage into or degeneration of the central nervous centers.

Convulsions may occur of epileptiform character, due to interference with the circulation of the brain.

Irregularities of gait, due to interference with the functions of the spinal cord, occur. Dementia may be the final stage, owing to degeneration of the brain.

Cardiac symptoms are common when the cardiac arteries are badly diseased.

In advancing arteriosclerosis there frequently occurs late in life, a thickening of the cardiac valves, giving rise to insufficiency and all the signs and symptoms of insufficiency due to a value deficit from acute inflammation of the valves.

Irregular heart action, slight dyspnea, moderate edema, attacks of pulmonary edema with expectoration of bloody froth, are somewhat common.

Cardiac pain due to myocardial insufficiency from thickening of the coronary arteries is common. It causes attacks of angina pectoris and often sudden death.

Renal System.—Albuminuria, tube casts with high specific gravity urine are all evidences of arteriosclerosis affecting the kidney as it has affected other parts of the body, and is to be differentiated from the condition of advanced nephritis known as Bright's disease.

Abdominal pain due to sclerosis of the abdominal vessels, known as *angina abdominalis*, may closely resemble attacks of acute indigestion and are often so treated.

Cramps in the muscles of the legs are often the result of arteriosclerosis, but of course may be due to overaction of the muscles without any demonstrable lesion of the arteries.

Intermittent claudication is a painful condition of the muscles due to arteriosclerosis, so that the muscle is not adequately supplied with blood.

Conditions to be Differentiated

The symptoms due to arteriosclerosis of special organs must be separated from the same symptoms due to other diseases of these organs:

Cerebral apoplexy
Cerebral tumor
Convulsions and other nervous disturbances

Cardiac disease due to infections
Functional heart disease
Locomotor ataxia.

CEREBRAL APOPLEXY.

Cerebral apoplexy has as its primary symptom, loss of power in some peripheral groups of muscles: the face, arm, leg—any one or all of these. This may be simulated by the temporary loss of power due to arteriosclerosis. In apoplexy, however, the loss of power continues longer if there is a hemorrhage, if due to cerebral anemia, the result of arteriosclerosis.

In arteriosclerotic disturbances the interference of function is but temporary. In severe hemiplegia the spinal fluid is frequently bloody.

CEREBRAL TUMOR.

Cerebral tumor differs from cerebral anemia in the fact that all of the symptoms—headache, convulsions, loss of power, failure of special senses—are progressive, not fleeting as in arteriosclerosis.

CONVULSIONS.

Convulsions due to uremia may be recognized by the loss of function of the kidney, as shown by the phenolsulphonephthalein, but it must be remembered that a simple albuminuria with a few tube casts is not sufficient reason to make a diagnosis of nephritis.

CARDIAC DISEASE DUE TO INFECTIONS.

Cardiac disease, if due to infection, such as rheumatism, has usually a history of these diseases antedating the occurrence of arteriosclerosis. As a rule general arteriosclerosis is not present in cases before middle life. It must be remembered also that at times high blood pressure in cardiac disease is compensatory, and not due to a disease of the peripheral vessels. It requires a careful examination of the history to differentiate the two conditions, which are identical in symptoms.

FUNCTIONAL HEART DISEASE.

Functional heart conditions can be differentiated by the fact that they have a positive causative factor, such as fright, fever, overexhaustion. They are more common in the young when arteriosclerosis is rare.

LOCOMOTOR ATAXIA.

This condition may be simulated by arteriosclerosis of the vessels of the spinal cord; but in tabes there is the Argyll-Robertson pupil, loss of patellar reflexes, loss of sexual power and sexual desire. These are not present in sclerosis of the vessels.

Section VIII

Diseases of the Urinary System

1. Movable Kidney

In the majority of women the right kidney can be felt below the edge of the ribs on deep inspiration. A true wandering kidney, however, may occasionally be felt far below its normal position, as far over as the pylorus, or down in the pelvis.

There is more or less pain, often paroxysmal. This pain occurs in distinct attacks of renal colic (Dietl's crisis) due to kinks of the ureter, often followed by discharge of a large amount of urine, often by a dragging sensation with more or less severe symptoms of neurasthenia. At times the kidney is so painful and in such an abnormal position that it may be taken for a tumor of some other organ, such as the stomach. It often gives rise to symptoms of indigestion, frequency of urination and pain in the region of the kidney (not a Dietl's crisis) and bladder and may lead to errors in diagnosis. Often the kidney is larger than normal and, according to Kelly, there is usually dilatation of the pelvis. It may occasionally be found in the true pelvis.

Conditions to be Differentiated from Movable Kidney

Tumors of the kidney
Tumors of other organs
Stone in the kidney
Cystitis
Hydronephrosis
Tuberculosis of the kidney.

TUMORS OF THE KIDNEY.

In tumor of the kidney there are no attacks of severe pain from Dietl's crisis, they are more apt to have hematuria. As a rule a tumor which can be surely diagnosed is much larger than a floating kidney. It is not nearly so movable as the simple loose kidney.

TUMORS OF OTHER ORGANS.

Tumors of other organs may be mistaken for movable kidney; however, they are not so movable as a rule, though a cystic gall-bladder may have such an elongated duct that it may be moved to almost any portion of the abdomen. There is usually disturbance of the organ affected by a tumor. There are no attacks of Dietl's crisis.

STONE IN THE KIDNEY.

Stone in the kidney, with its excruciating paroxysmal pain, may be mistaken for floating kidney because of the crises occurring in the latter condition. But the x-ray will show a shadow and pus and blood in the urine. An examination by the cystoscope and catheterization of the ureters will help to clear up the diagnosis.

CYSTITIS.

Frequent urination as a reflex act, with recurring pain referred to the bladder, may cause a diagnosis of cystitis. Urine examinations showing no signs of inflammation of the bladder, cystoscopy and x-ray will make the diagnosis negative for cystitis.

HYDRONEPHROSIS.

Hydronephrosis is a frequent accompaniment of floating kidney, but when due to this cause it is intermittent. An obstructive hydronephrosis is felt as a tumor which may be cystic and is constantly present. There are other symptoms of obstruction of the ureter. Congenital cystic kidneys in adults are rare; they are usually bilateral.

TUBERCULOSIS OF THE KIDNEY.

Tuberculosis of the kidney may be excluded by the absence of bladder infection, inflammation about the mouth of the ureter, the negative cultures and animal inoculations.

2. Passive Congestion of the Kidneys

Cause.—This condition is the result of failing cardiac compensation.

Characteristic Symptoms.—It is characterized by albuminuria, the presence of tube casts in the urine and the passage of diminished amounts of urine.

Differentiation.—It is to be differentiated from NEPHRITIS. This may be difficult, but the diagnosis can best be arrived at by the history of the case and consideration of the function of the kidneys. In a nephritis there is a history of disturbed renal function before the onset of the cardiac decompensation. If the nephritis be of the interstitial type, the blood pressure will be high. It must be remembered that a high blood pressure may be the result of a failing cardiac compensation. Usually the amount of albumin is greater in parenchymatous nephritis than it is in the disturbance due to cardiac decompensation. In interstitial nephritis the specific gravity of the urine is lower than in congestion of the kidneys.

Rest in bed, with the administration of proper remedies, will quickly dissipate the renal condition if there is any improvement in the general condition at all. This is not so markedly the fact where the renal disturbance is primary.

The use of phenolsulphonephthalein is here of the greatest value. If the kidneys are involved by an active inflammatory condition, the excretion of the phthalein in the first two hours will be diminished to a greater extent than if the renal condition is due to cardiac decompensation.

In *active congestion of the kidneys,* the beginning of a nephritis, there is never the history of cardiac decompensation.

3. Anuria

Suppression of urine must be diagnosed from *retention.* Suppression means, of course, the failure of the kidneys to secrete urine. If none is secreted consequently there will be none in the bladder; therefore, the obvious method of differentiation is catheterization of the patient.

Differentiation.—The only mistake that can be made would be in case of an accident in which the bladder is ruptured, and will contain little or no urine. This would be so evident by the other signs, such as bloody urine and extravasation into the soft parts, that an error is scarcely possible.

The only other possible mistake would be a urinary fistula which kept the bladder empty. This also could be easily differentiated by the history of the case and the presence of urine in other than normal positions.

4. Hematuria

Blood in the urine indicates some lesion in the genito-urinary tract which allows urine to be mixed with blood.

Differentiation.—The only mistake that might be made is the admixture of blood coming from the genital track in woman or hemorrhoids in man or woman.

In true hematuria the blood may come from the urethra, from the bladder, the ureters or from the kidneys.

Blood which comes from the urethra precedes the urine, or the very first flow of urine contains blood. If the bleeding point is in the posterior part of the urethra, the blood may immediately follow the urine; however, *blood which follows the urine* as a rule comes either from the bladder itself or from the ureter or kidney. *Blood which comes* from the kidney if in small amounts, as it is in scarlatinal nephritis, causes a *smoky* urine.

This may be represented in the following way:

From anterior urethra—Blood precedes urine or bright blood is mixed with it.

From posterior urethra or bladder—Bright bloody urine, smoky urine, blood after urine, or admixture of blood and urine.

From kidney—Smoky urine, blood in clots, sometimes tube casts.

Blood may occur in the urine in such small quantities that it gives no abnormal color to that fluid. This can always be diagnosticated by the use of the microscope which will invariably show blood corpuscles in the urine. Albumin is always present in the urine when blood occurs and except when the blood is in the very minutest quantities it can be demonstrated by the usual clinical methods. Under the microscope it is conceivable that the blood cells might be mistaken for various large mononuclear vegetative organisms, such as yeast, or they might be mistaken for some collection of minute substances, such as urates.

Only proper instruction in the use of the microscope and its employment in everyday practice will prevent such a mistake. The writer has seen the pink deposits of urea which occur occasionally upon the diapers of children, mistaken for blood. However, the diagnosis is easily made. Urates will dissolve in warm water and give no color; blood will not. The microscope will at once make the diagnosis absolute.

Hemoglobinuria can at once be distinguished from blood in the urine by use of the microscope. There are no corpuscles in the urine of hemoglobinuria.

Uretheroscopy, cystoscopy and catheterization of the ureters will give positive information as to the source of the blood in bloody urine.

If the bleeding point is in the urethera it can be distinguished by examination of that tube. If it is in the bladder, cystoscopy will show the bleeding point.

If the blood comes from the kidney, bloody urine can be seen coming from one or the other ureter, and catheterization of the ureters will show the urine to be either clear or bloody; it will also show whether kidney is affected.

5. Hemoglobinuria

Definition.—Hemoglobinuria is the presence in the urine of the coloring matter of the blood, without the presence of the actual blood corpuscles.

Occurrence.—This may occur in certain diseases such as malaria, or in some of the hemolytic diseases such as purpura and as the result of poisoning by certain drugs, and occasionally by hemolysis caused by the transfusion of blood.

Differentiation.—There is a curious condition existing called PAROXYSMAL HEMOGLOBINURIA, which is of rare occurrence. The urine contains granular matter, broken down blood disks and albumin, in addition to the hemoglobin. The positive test is spectroscopic examination of the urine. This will show either two absorption bands of oxyhemoglobin, or thin absorption bands of methemoglobin, the one in red near *C* being characteristic.

This can be mistaken for little else than hemoglobinuria. The spectroscope, as stated above, will make a positive diagnosis. It might be simulated by artificially colored urine, used for the purpose of deception, but the microscopic appearance will not resemble it.

Hematoporphyrin is present in urine in consequence of the continued and excessive use of certain of the coal tar preparations, such as veronal, trional, sulphonal. This can be differentiated by the history of the case of those in the habit of using the drugs and by a spectroscopic examination. The urine in hematoporphyrinuria does not contain albumin or any of the microscopic elements and does not correspond to any of the tests for hemoglobinuria.

6. Albuminuria

Definition.—Albuminuria is the presence of serum albumin in the urine from any cause whatever.

Cause.—Usually it is the result of some inflammatory condition of the genito-urinary tract, urethritis, cystitis, pyonephritis, and nephritis; it may, however, be the result of the toxin of fevers acting upon the kidneys and causing an entirely temporary disturbance of the function of the kidney.

Chemical Tests.—Albumin is recognized by several chemical tests. The one most useful in everyday clinical work is the *heat and acid test.* The urine in a clean test tube is boiled, and to this are added a few drops of acetic acid. If albumin be present there is a precipitant varying from a mere cloud to a heavy flocculent precipitant, amounting sometimes to practical coagulation of the entire mass of urine boiled.

Another test is the overlying test, or *Heller's test with nitric acid.* Here a small clean tube is partially filled with pure nitric acid, the filtered urine is allowed carefully to run down the side of the test tube until it reaches the acid. There is then a distinct layer of acid below and urine above. At the junction of the liquid and acid there is a white ring due to coagulation of the albumin. In individuals taking any of the coal tar preparations or iodid of potassium, or those with bile in the urine and all with highly pigmented urine from normal causes, a dark clear ring is found dipping in the urine below the line of albumin.

These two tests are sufficient for ordinary clinical findings. Many other tests are described, but for practical purposes the above suffice.

Conditions to be Differentiated from Albuminuria

Albuminuria may be mistaken for:

Phosphaturia
Albumosuria.

PHOSPHATURIA.

In phosphaturia, in alkaline urine when boiled a cloud appears very similar to the cloud due to albumin, but the addition of a drop or two of diluted acetic acid immediately clears up the cloud. A urate cloud might be mistaken for albuminuria in using Heller's test—a cloud forms in ring shape, one-eighth or one-quarter of an inch above the line of the acid. It must be remembered that the albuminuria ring rests directly upon the acid line. If there is any question as to the character of this ring of urates, a very gentle heating of the tube above the acid will cause the disappearance of the ring. Prolonged examination of the urine will show the presence of either pus, blood, or tube casts in the vast majority of urines containing albumin.

ALBUMOSURIA.

Albumose is found in many febrile conditions. This, together with Bence-Jones proteid, must be differentiated from albumin.

Bence-Jones proteid occurs in cases of myeloma and certain cases of leukemia. The reader is referred to Barker's Diagnosis in this series, for all of the tests. The simplest is the fact that Bence-Jones proteid causes a cloudiness in urine when heated to 50° C. and is completely coagulated at 70° C. The cloudiness decreases as the urine is further heated, and disappears at about the boiling point. Secondary albumoses do not respond to the ordinary tests for serum albumin.

7. Bacteriuria

Bacteriuria is the result of a growth of some form of microscopic organism in the urine. In certain conditions, such as typhoid fever, it is a marked characteristic of the condition. Usually the condition is patent to the naked eye by a cloudy appearance of the urine due to the growth of the organism, but this may always be confirmed or refuted by the use of the miscroscope.

Differentiation.—Bacteriuria may be confused with PHOSPHATES, or urates in the urine.

In phosphaturia the cloud caused by boiling the urine will always be dissipated by the addition of acid, and the cloud due to urates will be dissipated by heating the urine. Bacteria do not disappear from the urine either on heating or by the addition of acid. The bacteria which do not give rise to cloudy urine can only be diagnosed by cultivation of the organism from the urine or by their discovery by the microscope.

8. Pyuria

Definition.—Pyuria is the presence of pus in the urine, usually causing cloudy urine.

Cause.—This may arise from any inflammatory condition of the genito-urinary tract.

Diagnosis.—It may always be diagnosed by the presence of albumin in the urine and by the discovery of numerous leukocytes under the microscope. No definite number of pus cells to the field constitutes pyuria, but more than half a dozen to a one-sixteenth objective field may be so designated.

The macroscopic appearance of the urine in pyuria may cause some confusion because urates, phosphates, mucus and fat in the urine cause a cloudy urine.

Urates frequently cause a cloudy urine. Here the urine is clear when first passed, but on cooling it at once becomes cloudy; this cloud will in turn disappear when the urine is again heated.

Pus is noticed in the urine when it is passed, and heating purulent urine causes it to become more cloudy. Under the microscope the deposit will be seen to be amorphous in character, with no pus cells present.

Phosphates may be the cause of cloudy urine when it is first passed, or the cloud may become apparent only when the urine is heated to beyond the normal body temperature. The cloud may at once be dissipated by the addition of acid.

Mucus and epithelium may cause cloudy urine, but they can at once be distinguished by the use of the microscope.

Urine which is full of *bacteria* may be quite as cloudy as purulent urine. It, however, will not contain albumin, and the use of the microscope will show the absence of pus cells.

9. Acetonuria

Occurrence.—Acetonuria occurs under various conditions. The disease in which it shows its most typical features is diabetes mellitus. Chemists now assure us that the presence of acetone itself is not the cause of the symptoms present.

Symptoms.—In diabetes mellitus the symptoms consist of curious sweet odor to the breath, dullness of intellect, nausea, vomiting, and restlessness. This same series of symptoms occurs especially in young children who are suddenly seized with vomiting, depression, rapid running pulse, with nothing either in the history, physical condition or symptoms to account for it. There is, however, the presence of acetone in the urine, which can be demonstrated by the ordinary tests of the urine for that substance.

Conditions to be Differentiated from Acetonuria

Acute indigestion
Acute tonsillitis.

ACUTE INDIGESTION.

This condition is to be differentiated from acute indigestion in children—the result of overeating. Here the history of the case, together with the presence of undigested food in the vomitus and the *absence* of acetone and diacetic acid in the urine, will make the difference. It must be remembered, however, that an attack of acetonuria can occur at the time of ingestion of improper food. This food may be vomited and yet not be the cause of the condition present.

ACUTE TONSILLITIS.

The condition may be separated also from acute tonsillitis. The same rational symptoms arise in that condition as in acetonuria, and there is frequently the same sweet odor to the breath. Examination of the fauces will show the red inflamed pillars and fauces, with occasional spots of exudate on the tonsils. There is absence of acetone in the urine.

10. Indicanuria

Occurrence.—This condition is frequently present in cases of protein decomposition, especially where this decomposition takes place in the alimentary tract.

Diagnosis.—Indicanuria can be diagnosticated by the addition of hydrochloric acid to freshly passed urine, and of a 1 per cent solution of potassium chlorate. If chloroform be now added, the chloroform will absorb the indigo and deposit it as a deep blue layer.

11. Chyluria

Definition.—Chyluria is the presence of chyle in the urine—a milky opaque urine, closely resembling milk. The same macroscopic appearance may occur where there is no true chyle present in the urine.

Occurrence and Diagnosis.—The parasitic form is found where there is a connection of the thoracic duct with the urinary tract, and is the result of filiariasis. It can be discovered by the microscope, which shows the presence of minute granules, and more rarely oil drops in the urine.

Osler describes a non-parasitic form of chyluria in which the urine looks like milk, and sometimes coagulates into a jellylike mass.

Conditions to be Differentiated from Chyluria

It may be mistaken for:

Phosphates
Urates
Bacteria
Lipuria.

All these conditions are the cause of milky urine. PHOSPHATES, however, are dissipated by making the urine acid, URATES by heating the urine, and BACTERIA can be discovered by the microscope.

In LIPURIA fat drops can be seen in the urine, and the patient is the subject of serious condition—fatty stage of chronic nephritis, phosphorus poisoning, etc.

12. Lithuria

Definition.—Lithuria is the presence of unusually large amounts of uric acid or urates in the urine.

Diagnosis.—It may be apparent to the naked eye by the "brickdust" deposit seen in the urine, or deposits of a granular red mass. The color when the deposit is due to urates and not to uric acid varies from a white to a pinkish color.

It may be mistaken for PUS, or for a deposit of PHOSPHATES, but examination with the microscope will show that in pus there is a presence of leukocytes, while in lithuria either uric acid or urates can be seen through the microscope (Plate 3, Fig. 4).

Chemically the conditions may be distinguished by the fact that urates disappear upon heating the urine, while both phosphates and pus will cause the urine to become cloudy on heating. The cloud which occurs when phosphatic urine is heated will disappear upon the addition of acid; acid has no effect on the urates. Acid applied to a cloudy urine due to pus will probably cause it to become more intensely cloudy.

MUCUS and EPITHELIAL CELLS can be distinguished by the microscope.

13. Phosphaturia

Phosphaturia can be diagnosed by the appearance of urine which is cloudy when voided and which becomes clear on the addition of acid, or which is clear at the time of passing, becoming cloudy upon heating. Such urine usually deposits crystals of the triple or amorphous urates upon standing (Plate 3, Fig. 4). It is usually alkaline in reaction.

Differentiation.—ALBUMINURIA does not show a cloud in unheated urine. When albuminous urine is boiled it becomes cloudy, and the addition of acid does not dissipate the cloudiness, or it may not become cloudy on boiling if it is alkaline, but the addition of acid will cause it to become cloudy.

PUS and BACTERIA may cause cloudy urine when the urine is passed, but the cloud will not disappear upon the addition of acid.

14. Uremia

Cause.—This is a toxic condition, the result of deficient or total loss of the function of the kidney.

Characteristic Symptoms.—*In its more acute and severe forms* it is characterized by dullness of the intellect or complete coma and frequently mania or convulsions. There is usually high blood pressure.

In its chronic and less severe forms, it has as its symptoms, vomiting, loss of memory, somnolence, headache, epigastric pain, and temporary loss of power of any portion of the musculature. Urinous odor of the breath may be present. It is the common ending of fatal cases of nephritis. The phenolsulphonephthalein excretion is always below normal in uremia. The nitrogen content of the blood is usually far above the normal point.

Headache, visual disturbances and vertigo are extremely common symptoms. Complete blindness often occurs as the result of retinal hemorrhages and consequent sclerotic changes. The pupils differ in different cases; a retinitis may be present.

Loss of appetite, epigastric pain, and vomiting are common symptoms.

High blood pressure is common, together with hypertrophy of the heart and other cardiovascular changes; pericarditis and cardiac decompensation often occur; asthmatic attacks and constant dyspnea are common.

Albuminuria of varying degree, high or low specific gravity of the urine and tube casts are almost constant accompaniments of the condition.

The symptoms present in acute uremia are (first) convulsions, varying in type from a severe epileptic form seizure to mere twitching of individual muscles. The severe forms of convulsions may terminate in death. If the attack passes off, the patient becomes somnolent and has pains over the body; he may not regain consciousness enough to distinguish the condition. Sometimes, but by no means always, the temperature is raised during and after a convulsion; marked hemiplegia may occur, exactly resembling the hemiplegia of cerebral hemorrhage or thrombus.

A case lately observed at the Episcopal Hospital was known to have chronic interstitial nephritis. A gradually developing left hemiplegia occurred, which ended in death exactly comparable to that occuring in cerebral apoplexy. A careful autopsy failed to show any brain lesion independent of marked atheroma of the cerebral vessels.

Conditions to be Differentiated from Uremia

Epilepsy or other convulsive or unconscious seizures
Organic brain lesions
Stokes-Adams syndrome
Toxemia of pregnancy
Strychnin poisoning.

EPILEPSY OR OTHER CONVULSIVE OR UNCONSCIOUS SEIZURES.

The convulsive seizure itself cannot be differentiated, except other symptoms than those of a convulsion be present.

Those due to epilepsy can be differentiated by the antecedent history of convulsive seizures occurring over many months or years, often beginning in childhood. It is often accompanied by cerebral degeneration in the later stages. The urine is normal.

Blindness due to other causes may be recognized by the examination of the eye and the absence of albuminuric retinitis.

Unconsciousness due to diabetic coma may be differentiated by the presence of sugar or acetone and diacetic acid in the urine and by the absence of the symptom air hunger—usually neither albumin nor tube casts are in the urine of diabetics.

Sunstroke has a high temperature, a history of extreme to high degrees of heat.

In *apoplexy* there is always paralysis; in uremia it is rare.

Spinal puncture in apoplexy usually shows a bloody spinal fluid.

Concussion of the brain has the history of injury.

Opium poisoning has small pupils, slow respirations and the history of opium taking.

In *alcoholism* the patient can usually be aroused, and there is the history of a drinking bout.

In *syncope* the patient is pale and the pulse feeble.

In all of these conditions except diabetes mellitus the urine is normal and the phenolsulphonephthalein test is normal.

Convulsions due to functional digestive disturbances can be differentiated by careful examination of the digestive tract, by the history of the ingestion of improper food, by the vomiting of this food, and by the normal urine.

Convulsions due to gastric crises can be distinguished by normal urine and by the signs of locomotor ataxia in the patient.

ORGANIC BRAIN LESIONS.

Hemiplegia due to tumor, apoplexy, cerebral thrombus, or cerebral embolism can be distinguished by the normal urine and the history of the case. As stated above, a hemiplegia coming on during the course of a chronic nephritis cannot be differentiated except by autopsy in certain cases.

STOKES-ADAMS SYNDROME.

Stokes-Adams syndrome is a convulsive seizure accompanying a heart block. The ventricle beats much more slowly than the auricle; indeed the pulse at the wrist may entirely cease.

This is not the case in uremia, where the pulse is rapid and feeble.

The urine may be albuminous.

TOXEMIA OF PREGNANCY.

Pregnancy may be accompanied by a nephritis, but the pregnant state, the high blood pressure beginning with the pregnancy will influence the diagnosis.

Toxemia of pregnancy often has nephritis as a result. Here the pregnant state, the gradual rise of blood pressure and albumin before the appearance of tube casts are of import in the diagnosis. In this condition the epigastric pain so common as an early symptom of toxemia of pregnancy must be carefully differentiated from pain due to other causes.

STRYCHNIN POISONING.

In this condition the convulsions are *tonic*, not clonic, in character. The individual's intellect is clear. The convulsions may occur instantly and the individual die in the attack. There is the history of ingestion of the poison. The reflexes are increased.

The urine and the phenolsulphonephthalein tests are negative.

15. Acute Nephritis

Causes.—The commonest causes of nephritis are infections and intoxications.

Morbid Anatomy.—In almost every case of nephritis both the parenchymatous and interstitial tissues are involved; this is especially the case in the chronic forms. In the acute form, while the interstitial tissue is occasionally involved, the commonest condition is that of the involvement of the uriniferous tubules and glomeruli.

Characteristic Features.—Acute nephritis is characterized by sudden onset, frequently fever, great diminution in amount of urine or even total suppression. The urine which is excreted contains a large amount of albumin, frequently blood, leukocytes and many dark granular tube casts and blood casts (Plate 3, Fig. 3). Edema is a prominent symptom; it is often the first to attract the notice of the patient. Headache and dimness of vision are other prominent symptoms. Occasionally a convulsion, which is the expression of a uremia, is the first symptom to manifest itself.

Conditions to be Differentiated from Acute Nephritis

This condition might be mistaken for:

Simple retention of urine
Inflammation of the urinary tract exclusive of the kidney
Hematuria, not due to a renal lesion
Heart disease
Edema due to other conditions
Chronic nephritis
Cyclic albuminuria.

Retention of Urine.

This can be distinguished from the suppression due to nephritis by the simple use of the catheter. The urine will be found in the bladder; while it may be albuminous from some accompanying local trouble, it will not contain tube casts or other renal elements unless there is a nephritis plus retention.

Inflammation of the Urinary Tract Exclusive of the Kidney.

Purulent inflammation of the genito-urinary tract, excluding the kidney, is accompanied by albuminous urine, but the albumin will be found to be the result of pus in the urine. There are no tube casts or other renal elements; there is no edema, no heart complications or other signs of systemic condition relative to the kidneys. Instrumental examination of the bladder and ureters will show the local inflammation.

Hematuria, Not Due to a Renal Lesion.

Hematuria also gives albuminous urine, but the albumin is found to be the result of blood *without casts.* If the blood comes from the urethra it usually precedes the flow of urine; if it comes from the bladder it may be mixed with urine.

Heart Disease.

The condition might be mistaken for heart disease because of the cardiac weakness and edema often accompanying it, but the comparative freedom from dilatation of the heart with the previous history and the absence of murmurs of organic character will make the differential diagnosis. The albumin and tube casts which accompany heart disease are only slight in amount and there is not so much diminution of the urinary output as there is in acute nephritis.

Edema Due to Other Conditions.

The edema of anemia is not accompanied by urinary findings of nephritis.

Edema due to the use of arsenic might be mistaken for acute nephritis, but in the former there will be no abnormal urinary findings, and there is apt to be a gastro-enteritis accompanying arsenical poisoning.

Chronic Nephritis.

In this condition there is a history of long standing; suppression of urine is not a symptom; the tube casts are either light granular or fatty. The blood pressure is apt to be high.

A convulsion as the result of some other infectious disease, or a reflex convulsion, might be mistaken for the convulsion of the uremia of acute nephritis. Here, however, there is fever; the signs of the disease, sore throat, a rash, etc., will be present. The urinary findings are entirely normal in such conditions.

Every infection of serious degree is accompanied by albuminuria, and sometimes with tube casts. This condition must be looked upon simply as an effect of the circulatory poison upon the kidney and not as a primary nephritis.

Unconsciousness due to epilepsy, cerebral hemorrhage, injuries to the brain, alcoholism, diabetes, sunstroke, opium poisoning and the consciousness due to cardiovascular changes must not be mistaken for uremia due to acute nephritis. In these conditions these several symptoms will be present. The urine differs from that of acute nephritis.

Cyclic Albuminuria.

There is also a condition of cyclic albuminuria, for which it might also be mistaken, in which albumin is present at intervals. Here there are none of the kidney elements constantly present in nephritis, and the albumin often entirely disappears without apparent reason.

16. Chronic Nephritis

Etiology.—Chronic nephritis may be the result of an acute attack the lesions of which have caused permanent damage to the kidney, or it may be the result of some slowly acting systemic poison; this poison may

PLATE IV

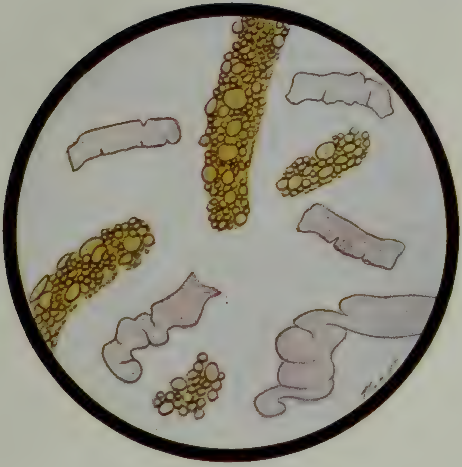


Fig. 1.

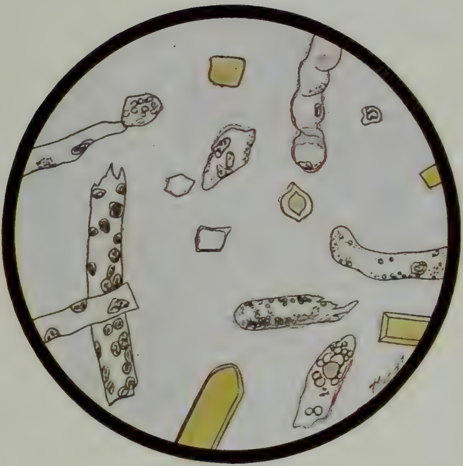


Fig. 2.

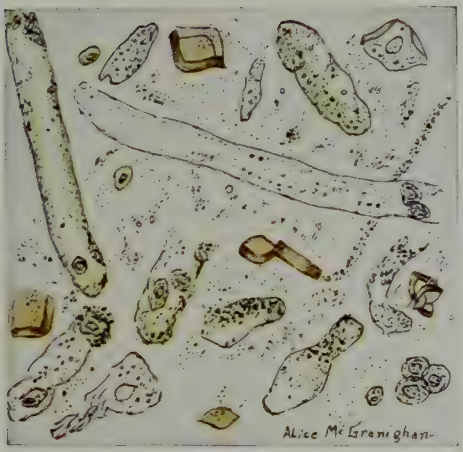


Fig. 3.

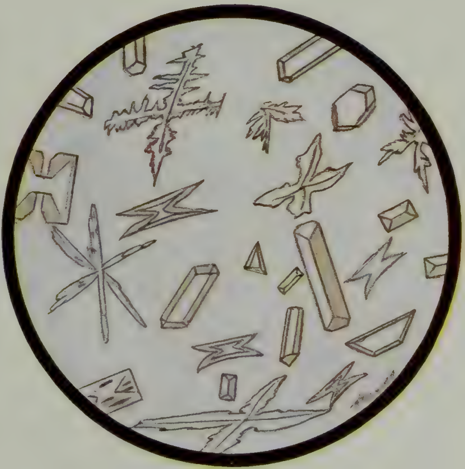


Fig. 4.

Fig. 1.—Fatty and Waxy Casts from a Case of Chronic Nephritis. (Adapted and Colored by Rieder and Delépine.)

Fig. 2.—Hyaline and Granular Tube Casts from a Case of Chronic Nephritis. (Adapted and Colored by Rieder and Delépine.)

Fig. 3.—Acute Toxic Nephritis. Tube Casts, Uric Acid Crystals, Amorphous Urates. (Rieder's Atlas, Edition 1898.)

Fig. 4.—Triple Phosphates in Urine. (Adapted and Colored by Rieder and Delépine.)

have its effect principally upon the parenchyma of the kidney, or it may attack the vessels of the kidney simultaneously with the general vascular system. The kidney in the human subject is so constructed that a considerable amount of secreting tissue may be destroyed before its function is enough diminished to give rise to general symptoms. Thus we frequently, for many years, have albumin and tube casts and possibly high blood pressure as the only symptoms.

Such cases demand diagnosis and treatment in order that the patient's mode of living may be so regulated that his life may be made comfortable and be as prolonged as is consistent with his condition. Other cases are more advanced and may attract attention by indigestion, headache, anemia, small amounts of urine containing much albumin and many tube casts or large amounts of urine containing few tube casts. There may also be polyuria with much albumin and few casts. There may be symptoms of chronic uremia.

In individuals with *interstitial nephritis* the vessels of the kidney are principally involved and the renal change is principally interstitial in character. The first symptoms may be dyspnea, slight pretibial edema, vertigo and headache as symptoms, the general physical examination showing cardiac hypertrophy, high blood pressure, rather abundant urine of low specific gravity, containing a few tube casts usually light granular in character. This condition, too, demands differentiation and diagnosis that the patient may enjoy as much of his allotted term of life as possible. Later, as the kidney substance becomes more impaired, the renal symptoms come into the foreground characterized by chronic uremia, edema, etc.

The degree of kidney insufficiency in both of these conditions is well indicated by the use of the phenolsulphonephthalein test. Frequently as the case progresses the phthalein output diminishes gradually and a good idea of the prognosis may thus be obtained. Constantly these two great types of cases merge, both because of the increase in inflammation in the parenchymatous tissue in the one and the increase of interstitial change in the other. Border line cases occur in which it is impossible to determine whether the parenchyma or interstitial change is in the ascendant.

Chronic forms of nephritis may broadly be divided into the parenchymatous and interstitial varieties. As said before, these forms merge, and pathologically, interstitial and parenchymatous changes are found in practically all kidneys the subject of chronic disease, in some a preponderance of parenchymatous changes, in others of interstitial changes. Clinically, however, these two forms are fairly well distinguished.

The individual with *parenchymatous nephritis* passes an amount of urine only slightly above normal, or perhaps below normal. It is of about normal specific gravity, contains much albumin and many tube casts. There is severe anemia, a tendency to edema and weak cardiac action, the blood pressure often being only slightly raised.

Differentiation.—The first condition must be differentiated from:

Albuminuria which accompanies genito-urinary purulent conditions.

The anasarca and anemia which are part of the symptomatology of the primary anemias.

Heart disease which has brought about such secondary change in the kidneys that the urinary findings may simulate a primary nephritis.

Purulent inflammation of the kidneys or any other form of genito-urinary inflammation which is accompanied by pus and blood in the urine whether an actual nephritis is present or not.

In these cases there is generally a faulty normal function of the kidney as shown by the phthalein test. Tube casts may or may not be present. Careful cystoscopic examination and catheterization will show the local lesion. There is pus in the urine. There are no cardiovascular changes, which are common in nephritis, and the blood pressure is not high.

Primary Anemia, Pernicious Anemia, Leukemia and Hodgkin's Disease.

This condition may be accompanied by general anasarca and rather profuse urinary output; the blood examination will always show the primary disease. A red blood count below or approaching 1,000,000 red cells to the cm., with poikilocytes and nucleated reds, will practically always mean a primary anemia, or perhaps a severe anemia the result of dibothriocephalus infection or of syphilis.

Increase of the white cells of a mixed character, or of the lymphocytes particularly, will mean leukemia. Here, too, enlargement of the spleen with general glandular enlargement will mean leukemia. There is no albumin or tube casts in these conditions.

Hodgkin's disease may be differentiated by means of enlargement of the glands, the presence of the typical change in a section of the gland and a more or less continued intermittent fever. The blood picture changes with the progress of the disease.

Secondary anemia which may have anasarca and cardiac dilatation as characteristics is indicated by its peculiar characters. There may be a few tube casts and a small amount of albumin in the urine of these conditions, but the function of the kidneys as indicated by the phthalein test is not altered.

Then in all these conditions the betterment of the patient as the anemia decreases at once clears up any urinary signs.

Cardiac Decompensation.

Cardiac decompensation is almost constantly accompanied by decreased urinary output, albuminuria and tube casts. In these cases, however, there is generally the history of long-standing cardiac disease, or the physical examination will show the heart much dilated, with or without murmurs, with failure in the quality of the heart sounds. Here rest and appropriate remedies and direction to correction of cardiac decompensation will show a total or almost complete clearing up of the urine.

The phthalein test, even in the presence of severe renal involvement, shows a good function of the kidney as compared with that present in true nephritis. If the phthalein output is decreased, it will become nearer normal as the heart condition improves.

The true INTERSTITIAL FORMS OF NEPHRITIS are thought to be a primary *renal condition.* They must be carefully differentiated from those conditions where the main symptoms are due to renal involvement as a part only, and generally a small part of the diseased state.

In these latter cases rest in bed with careful regulation of diet and later exercise with hygienic and dietetic treatment together with remedies for the heart, will so clear up the case that the renal condition, while still a part, will easily be seen to be only a small part of the symptomatic picture.

It is in cases with albuminuria and casts that Cabot has shown that the mere presence of albumin and casts is not alone a true guide for a diagnosis of nephritis. In his conclusions, as follows, he seems to indicate that careful urinary examinations are useless: "The important points in examination of urine are its color, its amount, and its specific gravity."

Careful urinary examination, on the contrary, *is imperative in every case;* nothing is more neglected. While we make mistakes, the man who is painstaking in a complete urinary examination, in his history and in his physical examination, will make many less blunders than he who omits any one of the three of these necessary elements.

17. Primary Syphilitic Nephritis

It is frequently a question of importance to tell whether a nephritis which is present in the course of an acute or chronic syphilis, is due to the syphilis, or whether it is the result of another infection, and is a complication of syphilis.

Munk has shown recently (Berl. klin. Wchnschr., 1913, 2-1416) that the urinary sediment in ordinary acute nephritis contains fatty material when viewed through a microscope fitted with Mikel's prisms, whereas the sediment present in a nephritis due to syphilis contains lipoid substances when examined in the same way.

This test is of value in deciding upon the form of treatment, whether neosalvarsan may be used or not, and is the only certain method of differentiation, because the Wassermann test, for instance, means simply that the patient is syphilitic and not that the nephritis is necessarily syphilitic.

18. Amyloid Disease—Lardaceous Disease

(*Waxy Degeneration*)

This is an organic change due to long-standing suppuration and is characterized by formation in the various tissues of a substance called lardacein.

The patient with long-standing suppuration of a bone—as in Pott's disease, hip disease or in advanced syphilis—gradually grows pale, the tissues become edematous, the urine contains albumin with polyuria and there is diarrhea. The spleen and liver become enlarged. The characteristic of the urine is low specific gravity with a large amount of albumin, containing hyaline and waxy casts. When the intestines are involved, there is diarrhea.

Conditions to be Differentiated from Amyloid Disease

This condition can be mistaken for:

Parenchymatous nephritis
Cirrhosis of the liver
Urinary disturbance due to cardiac decompensation.

PARENCHYMATOUS NEPHRITIS.

Parenchymatous nephritis, by reason of the general anasarca and the large amount of albumin in the urine, might be mistaken for amyloid disease, but in the latter there is polyuria. Then, too, the casts are of different character; in amyloid disease there are few casts, and these are either hyaline or amyloid. There is also the absence of a history of suppuration or syphilis. The liver and spleen are rarely enlarged.

CIRRHOSIS OF THE LIVER.

Cirrhosis of the liver may have an enlarged liver as well as a large spleen; there is, however, a marked tendency to ascites which is not present as a rule in amyloid disease. There is rarely nephritis; when it occurs the albumin is much less abundant. There is the history of alcoholism but not of long-standing suppuration. The patient is not so markedly edematous as he is in amyloid disease.

URINARY DISTURBANCE DUE TO CARDIAC DECOMPENSATION.

Albuminuria due to this condition is detected by the presence of a heart lesion which is manifestly organic in character. There is also the absence of a cause, such as suppuration or syphilis.

19. Pyelitis

This condition, whether due to nephrolithiasis or some infection independent of lithiasis, is characterized by purulent urine. The urine contains pus curiously free from mucus and it also contains renal epithelium.

Frequently there is pain over the kidney affected. Occasionally there is an intermittence of the flow of pus, and often intermittent fever.

Differentiation.

The condition may be confused with:

URETHRITIS
CYSTITIS
RENAL SUPPURATION.

Careful examination of the urethra will show the presence of an ulcerated area along that tube if the pus comes from the ureter. Examination of the bladder with a cystoscope will show the presence of an inflamed bladder wall, if the condition is due to inflammation of that organ. Examination of the mouth of the ureter at the time urine is discharged into the bladder will show purulent urine coming from the affected side if the seat of inflammation is the pelvis of the kidney or in the kidney itself.

If there is doubt whether the urine comes from a pyelonephritis or from a simple pyelitis, collargol may be injected through a catheterized ureter and an x-ray picture taken.

The character of the inflammation may be differentiated by the following procedures:

An x-ray will show the absence or the presence of a calculus; it will also show the presence of cavities in the kidney if collargol be first injected; cultivation of the organism from the urine of the affected side will show the character of the infecting organism; animal inoculations will cause tuberculosis in the animal if the infection is due to tuberculosis.

20. Hydronephrosis

Hydronephrosis, almost without exception, gives rise to a renal tumor in the region of the kidney which frequently disappears temporarily. The disappearance is accompanied by the flow of a large amount of urine.

Conditions to be Differentiated from Hydronephrosis

This tumor must be differentiated from:

A new growth—a hypernephroma, a sarcoma, or a carcinoma
A congenital cystic kidney

A single large kidney
A tuberculous kidney
A new growth not connected with the kidney
Pancreatic cysts.

The history is somewhat typical, as a hydronephrosis is the result of a blocking of the ureter either by a stone or a kink or a congenital stricture in the ureter. There is the history of renal calculus, or of floating kidney with the characteristic symptoms of these conditions. Catheterization of the ureters will show a blocking of the ureter on the affected side.

New Growth.

A new growth is solid; a hydronephrosis is likely to be fluctuating. There is usually failure of the general condition of the patient in new growth which is not likely to occur in a hydronephrosis. If it is a case of a new growth there are likely to be many other symptoms than those of tumor, though a hemorrhage may take place; then, too, the size is likely to greatly exceed that of a hydronephrosis. A new growth gradually increases in size.

A new growth of the kidney is often accompanied by hemorrhage, the hemorrhage taking the form of a cast of the ureter. This cast is often of huge size, the large diameter being the result of overdistention of the ureter.

Congenital Cystic Kidney.

Congenital cystic kidneys are apt to be bilateral; their function is usually about normal until some sudden accident causes them to cease. When the mass is unilateral the tumor does not vary in size.

Single Large Kidney.

A single large kidney can only be diagnosed by assurance of the absence of the other kidney; this may be done first by catheterization of the ureters. Usually a congenital absence of the kidney means also an absence of the ureter on that side. This might be simulated by the enlargement of the remaining kidney after the destruction of one by disease. In this condition frequently the ureter can be demonstrated on the affected side by the use of a ureteral catheter and x-ray.

Tuberculous Kidney.

Tuberculosis of the kidney may cause enlargement and degeneration of the kidneys, but here the urine can be taken from the diseased side and examination will show the presence of pus and frequently also the presence of tubercle bacilli.

New Growth Not Connected with the Kidney.

A tumor due to a movable kidney can be differentiated by its solid character, its moderate enlargement, its movable character and the ability to replace the kidney under the edge of the ribs. A tumor other than a renal one might resemble a hydronephrosis. Usually, however, the renal tumor has the colon either in front of it or the colon is pushed down. Often there is some difficulty in distinguishing between an enlarged kidney on the left side and an enlarged spleen. A spleen, however, can usually be distinguished by the fact that it extends further up under the ribs, a notch can be distinguished, and the edge is sharp instead of rounded. Catheterization of the ureters with an opaque catheterization and an x-ray taken will show whether the catheter enters the tumor.

A cystic gall-bladder which is greatly enlarged may be mistaken for a kidney, but there is a difference in position: it is in front of the colon. There are no urinary symptoms, and it is frequently accompanied with inflammatory symptoms referable to the bile passages. Catheterization of the ureter will not show any lack of urinary flow, and an x-ray will possibly show the kidney in its normal position.

A pyloric tumor has been mistaken for a renal tumor. Here, however, there are distinct gastric symptoms, and use of gastric lavage, x-ray, and inflation of the stomach will show the tumor in the gastro-intestinal tract.

Pancreatic Cysts.

Pancreatic cysts may be mistaken for hydronephrosis, but for the following facts: the position is more central in the abdomen; there are no symptoms of renal disturbance; the tumor is often of greater size.

21. Nephrolithiasis

Characteristic Symptoms.—Stone in the kidney is characterized by two sets of symptoms. The *acute symptoms* of renal colic are severe agonizing pain with its point of maximum intensity in the region of the kidney and along the ureter, the pain being conducted into the bladder in the female and further in the penis or testicle in the male. This paroxysm is accompanied by frequent urination; the urine contains blood, leukocytes and sometimes tube casts when the irritation is long continued. The pain is so extreme that almost without exception morphin or even chloroform is needed to control the paroxysms. The kidney is often enlarged and tender. If the calculus passes into the bladder the pain ceases immediately.

A stone impacted in the kidney or ureter may give rise to the above symptoms of renal colic, but in addition there is more or less *constant* distress. The urine continually contains pus, blood, and other elements showing irritation of the kidney or pelvis. The use of an x-ray will show a shadow of the stone either in the kidney substance, in the pelvis or in the ureter. If this fails examination by catheterization of the ureter will show disturbance of the kidney function on the affected side. Later in the disease there may be emaciation or signs of suppurative disease which can usually be located in the kidney region. Ureteral catheterization or choloroscopy will show loss of function of the kidney on the affected side.

Conditions to be Differentiated from an Acute Attack of Nephrolithiasis

The acute attack may be mistaken for either:

Dietl's crisis
Obstruction of the ureter
Appendicitis
Acute cholecystitis.

DIETL'S CRISIS.

Dietl's crisis accompanies floating kidney. The kidney can usually be demonstrated as a more or less hard, elastic mass, tender to the touch and movable. With the cessation of the attack the kidney can still be felt; it is not tender. An x-ray will show the kidney but no shadow of a stone.

OBSTRUCTION OF THE URETER.

Obstruction of the ureter other than by stone may occur in pregnancy or as a result of pressure from an abdominal tumor or involvement in a new growth. Here the pregnancy or new growth can be demonstrated in the abdomen or pelvis. Urinary examination will not show blood.

APPENDICITIS.

Appendicitis may resemble renal colic in the acuteness of the pain. Usually, however, there is fever, local tenderness over McBurney's point, and absence of signs in the urine. Occasionally, however, as in a case reported by the author, there is both blood and pus in the urine, but presence of leukocytosis will make the diagnosis in favor of appendicitis.

ACUTE CHOLECYSTITIS.

Cholelithiasis, or cholecystitis, might be mistaken for renal colic, but the position of the pain differs; there is tenderness and pain over the gall-bladder; often there is fever and leukocytosis and jaundice.

Conditions to be Differentiated from Impacted Stone

An impacted stone may be mistaken for:
Tuberculosis of the kidney
Caries or arthritis of the spine
Perinephritic abscess
Chronic gall-bladder disease
Stone in the bladder
Stricture of the urethra
Lumbago.

TUBERCULOSIS OF THE KIDNEY.

Tuberculosis of the kidney may be secondary to tuberculosis in other portions of the body. If that is the case, signs in the lungs or other organs will help to point to a tubercular origin. The greatest help is an x-ray and catheterization of the ureters which will show a shadow if there be a stone and none if there be tuberculosis. Examination of the urine obtained by the urethral catheter will show tubercle bacilli, and animal inoculation with the urine will cause tuberculosis in the animal if the condition is due to tuberculosis.

CARIES OR ARTHRITIS OF THE SPINE.

Caries or arthritis of the spine may give rise to pain which will simulate the pain either of acute renal colic or the pain accompanying the impaction of a stone. However, there is limitation of motion of the spine of a particularly spasmodic character, the pain is aggravated on motion and at night but is more or less continuous. Examination of the spinal column will show tenderness over one or the other vertebrae. An x-ray examination of the spinal column will show erosin and arthritis of the vertebrae and will not show the presence of a shadow in the kidney or the ureter.

PERINEPHRITIC ABSCESS.

Perinephritic abscess is accompanied by tenderness and pain in the loin, with fever and leukocytosis. When on the right side, it is usually the result of an appendicitis which may have been latent, there may be the history of a previous appendicitis of a peripheral infection not connected with the kidney, or it may be the result of a long-continued renal lesion. The urine is normal unless the perinephritic abscess is the result of a kidney infection. Under the latter condition the urine will contain blood and pus.

CHRONIC GALL-BLADDER DISEASE.

In gall-bladder disease there may be pain with symptoms of fever, emaciation and leukocytosis, but the physical signs and the urinary findings differ in the two diseases.

STONE IN THE BLADDER.

In stone in the bladder the first symptoms are usually frequent urination, dysuria, and other signs of cystitis. There is blood and pus, but usually much mucus. Examination of the bladder with a stone sound will decide the presence of a calculus in the bladder. An x-ray will often show the stone in the bladder.

STRICTURE OF THE URETHRA.

A urethral stricture, even of moderate lumen, may give rise to reflex symptoms resembling a stone, but the x-ray and urinary findings differ entirely in the two conditions. The stricture will be demonstrated by proper instrumentation.

LUMBAGO.

Lumbago is the result of a myalgia affecting the erector spinæ group. There is marked local tenderness and movement is difficult. The x-ray picture will not show a stone. Another characteristic of lumbago is that the pain is usually lower than it is in stone in the kidney.

22. Tumors of the Kidney

Etiology.—New growths of this organ are chiefly hypernephromata, various names having been given by pathologists to tumors of the same character, but a tumor may be carcinoma or sarcoma; it may be embryonic.

Hypernephromata are believed to have their origin in adrenal tissue, occurring in abnormal positions (adrenal rests). The tumor may be very small or reach a tremendous size, occupying the greater part of the abdominal cavity; between these two extremities there is every gradation. Except for the very evident tremor they may be symptomless. There may be metastasis to almost any portion of the body and these tumors may be the first symptoms of the disease. A patient was recently seen with a tumor reaching from the seventh rib to a hand's breadth below the umbilicus. There had been a slight loss of weight, but besides that and a very slight dysuria there were no symptoms (Fig. 94).

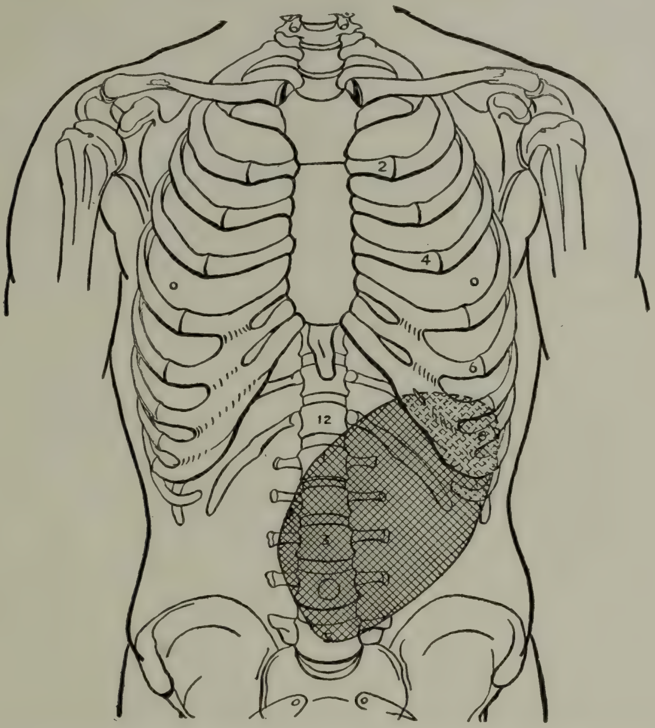


Fig. 94.—Hypernephroma. The Cut Shows the Area Over Which the Kidney Could Be Felt and Percussed. (Original Observation).

Symptoms.—The usual symptoms are hematuria and pain and the presence of tumor.

Conditions to be Differentiated from Tumors of the Kidney

The condition must be diagnosed from other states causing hematuria and pain or tumor. Among these are tuberculosis, nephrolithiasis, hydronephrosis, congenitally large cystic kidney, hypertrophic kidney, perinephritic abscess, tumors of spleen and other tumors of the kidney.

Tuberculosis of the Kidney.

Tuberculosis of the kidney may have hematuria as a symptom; the kidney may be large or small; there is usually pyuria. Examination of the bladder will show irritation of the mouth of the ureters, perhaps tuberculosis of the bladder itself; examination of the urine by microscopical examination may show acid-fast bacilli which may be confirmed as tubercle bacilli by animal inoculations.

It must be remembered that acid-fast bacilli in the urine may be smegma bacilli. The differentiation can be made certain by cultivation and by animal inoculation. There may be tuberculosis in other organs. Catheterization of the ureters, collargol injections and x-ray may show tuberculous foci in the kidney. The diagnosis is of the highest importance when the kidney is small; an extremely large renal tumor is rarely tuberculous. Gibbon (personal communication) suggests the surgical exposure of both kidneys when doubt exists as to whether one or both kidneys are diseased—probably a severe but extremely important procedure.

Stone in the Kidney or Bladder.

Stone in the kidney or bladder often has characteristic paroxysmal pain with pus and urine in the kidney. An x-ray will discover the stone. Cystoscopic examination will show the condition of the bladder and the stone in the bladder if one exists there. Reflex pain from stone in the bladder may simulate pain originating in the kidneys.

Hydronephrosis.

Hydronephrosis may cause a very large tumor; usually it is cystic and can be made out by palpation. The varying size of the tumor is almost certainly due to intermittent filling of the distended pelvis of the kidney.

Hematuria.

Hematuria, so-called essential hematuria, hemorrhage due to beginning nephritis and to purpura, can be differentiated by the absence of all of the signs of the above conditions and by tube casts in the urine in beginning nephritis, or by the presence of bleeding from other organs in purpura.

Carcinoma.

Carcinoma is of shorter duration than is a hypernephroma, death usually occurring within a year—often with the beginning of the symptoms. There is hematuria.

Sarcoma.

Sarcoma are more frequent in children, but it is probably impossible to make a differentiation from the more malignant hypernephroma.

Tumors of the Spleen.

Tumors of the spleen are always felt on the left side. They can usually be located by the shape of the organ; the edge is sharp and frequently a notch can be felt. Examination of the blood showing leukemia will decide the tumor as splenic; exploratory incision can be done in doubtful cases. Hematuria does not occur and pain is less likely to be present than in tumors of the kidney.

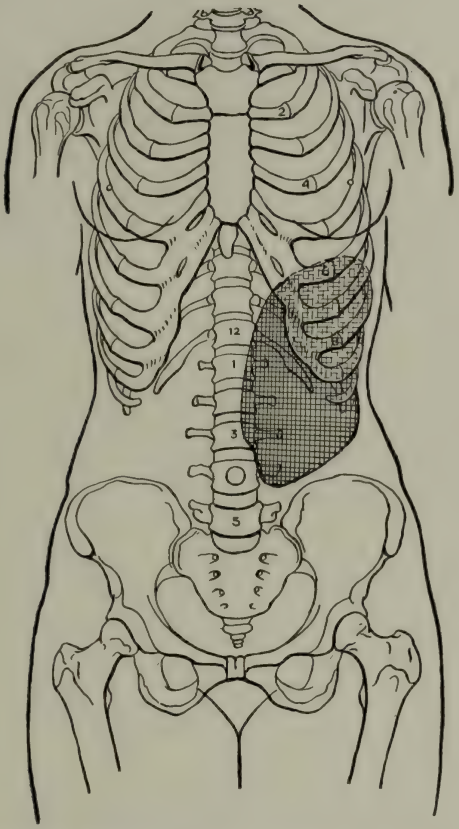


Fig. 95.—Area of Spleen in a Case of Myelogenous Leukemia. (Kindness of Dr. Petty.)

A Floating Kidney.

Floating kidney is often enlarged and painful, but the normal shape of the kidney can be outlined.

According to Osler, a condition of dilatation and hypertrophy of the bladder accompanying congenital defect of the abdominal muscles may be mistaken for the condition. The catheter will make the diagnosis.

Congenitally Large Cystic Kidney.

Cystic degeneration (congenital) of the kidney may at times be so great as to complicate labor. When this condition is recognized during life, the kidneys, as a rule, gradually increase in size, having the appearance of bilateral tumors; the renal function fails and the patient dies of uremic symptoms.

A remarkable case of the kind occurred in the practice of the writer lately, in which the tumor was first mistaken for an ovarian cyst, but careful examination soon discovered the tumor to be bilateral and uremic symptoms prevented a contemplated operation.

23. Perinephritic Abscess

Definition.—Perinephritic abscess is a collection of pus in the perirenal tissue.

Etiology.—Its cause may be an infected kidney, a suppurating appendix or other abdominal organ, or caries of the spine; it may be part of a general infection or it may be due to a wound in the kidney region.

Symptoms.—Its symptoms are pain, tenderness and fullness over the renal region; there is fever of septic character and leukocytosis. It may follow years after an infected bladder, or it may occur after a kidney has been entirely destroyed by a suppurating process. It may be the symptom first recognized in the course of appendicitis.

Diagnosis.—It may be suspected in the course of a suppurating kidney, stone in the kidney, severe cystitis, appendicitis, or in the course of an infection—by the appearance of pain and tenderness with a tumor in the lumbar region. The urine is always normal unless there be a connection between the kidney or renal pelvis and the abscess.

Conditions to be Differentiated from Perinephritic Abscess

It may be confused with:

Caries of the spine
Hydronephrosis
Suppuration of the kidney
Appendicitis
Tumors of the kidney
Enlargement of the spleen.

Caries of the Spine.

Caries of the spine may cause great tenderness over the lumbar region, but fixation of the spine together with the long history of the case and an x-ray picture will make the diagnosis positive.

Hydronephrosis.

A large hydronephrosis may show fluctuation in the loin. There is local tenderness but no fever, no leukocytosis and no redness such as often occurs in perinephritic abscess.

Suppuration of the Kidney.

Suppuration of the kidney may give rise to all of the symptoms of a perinephritic abscess except the pointing of an abscess in the renal region, but usually there is pus in the urine, and catheterization of the ureter will show pus coming from that organ. There may be a pyonephrosis, however, plus a perinephritic abscess. Here the main diagnostic point must be the fluctuation in the region of the kidney.

Appendicitis.

Appendicitis, as stated above, may give rise to a perinephritic abscess on the right side, but occasionally the pain of appendicitis is reflected high up over the kidney region without an actual collection of pus being present. If the lesion is confined to the appendix there will be tenderness over the right iliac fossa, there will not be fullness and fluctuation over the region of the kidney.

Tumors of the Kidney.

Tumors of the kidney are solid to palpation; they do not fluctuate; the condition is not acute; there is no leukocytosis.

Enlargement of the Spleen.

Enlargement of the spleen may occur from many causes other than leukemia. The mere fact that there is no abnormality of the white cells of the blood, either in number or character, will prove that the enlargement is not due to any form of leukemia. A history of syphilis or of an anemia with symptoms of cirrhosis of the liver argues in favor of a spleen. There is no tenderness or fluctuation over the spleen.

Section IX

Diseases of the Blood

General Considerations

The blood should be considered as much a tissue as any other portion of the body. It is affected, so far as we at present know, by disease in the blood-making organs themselves, by general conditions which affect the blood-making organs, and also by other conditions which affect the blood itself, independently of the blood-making organs. It is a well-known fact that blood consists of plasma and serum. The plasma contains fibrin, white and red corpuscles and blood platelets.

The disease which affects the blood-making organs, affects the various constituents of the blood, and the diagnosis of the existing condition can usually be made by microscopical examination of the blood itself.

1. Hemorrhage

Hemorrhage may be the result of a separation of a large blood vessel, or it may be the result of a continuous exudation from numerous small blood vessels, or the result of capillary oozing. The hemorrhage may be either external or internal.

External Hemorrhage.—External hemorrhage is the result of a laceration of a blood vessel which is near the surface of the body. The appearance of blood at the site of a laceration is of course a pathognomonic symptom. Accompanying this is rapidly increasing pulse faintness which is preceded by vertigo, syncope and low blood pressure.

Internal Hemorrhage.—There are the same general symptoms of hemorrhage whether it occurs superficially or internally. For instance, the first symptom of bleeding from the stomach may often be faintness, syncope, feeble rapid pulse and anemia; later there will be vomiting of blood and passage of blood from the bowels.

The rupture of an internal aneurism, which is usually fatal because of the hemorrhage, gives rise to exactly these same symptoms. Capillary bleeding or loss of blood from erosion of a small vessel, may at first give no symptoms except the appearance of blood at the seat of the lesion. For example: blood with a stool, hemorrhoids, continuous bleeding from the uterine cavity, blood from the gums in scurvy or hemophilia; but the symptoms noticed are paleness of the individual, followed by rapid pulse, dyspnea, palpitation of the heart and syncope. The condition of the blood in all of these instances is as follows:

Large Massive Hemorrhage.—In the large massive hemorrhage, whether it be internal or external, the number of corpuscles is reduced, the hemoglobin reduced to an equal degree, which is more or less the case where the hemorrhage is slight and capillary in character. Here, however, while both hemoglobin and red corpuscles are diminished, the hemoglobin is reduced proportionately more than the red corpuscles; in other words, there is secondary anemia. There is practically no change in the white corpuscles though in massive hemorrhages they may perhaps first rise and then be reduced below the normal number.

Conditions to be Differentiated from Hemorrhage

Hemorrhage may be confused with:

Fright
Faintness
Cardiac disease
Vertigo due to disease other than bleeding
Cerebral anemia
Shock.

FRIGHT.

Fright may cause the pulse to become rapid, the heart to palpitate, the face to become pale and may even cause unconsciousness.

The absence of any disease which might cause bleeding, the absence of visible blood, of cardiac disease, of gastro-intestinal disturbance, such as ulcer of the stomach, typhoid fever, uterine disease and cirrhosis of the liver—the absence of all these will make the diagnosis positive against hemorrhage. Examination will show normal blood in cases of fright.

CARDIAC DISEASE.

Cardiac disease, especially sclerosis of the coronary arteries, may cause faintness, paleness, irregular weak heart and all of the symptoms of hemorrhage. The diagnosis of this condition as the cause of the symptoms must depend upon the history of the case and the physical examination of the patient. It must be remembered that in these conditions of cardiac disease with great dilatation, there may be no audible murmur; hence dependence must not be placed upon the absence of a murmur. The hemoglobin and red cells will not be decreased.

VERTIGO DUE TO DISEASE OTHER THAN BLEEDING.

Sclerosis of the cerebral vessels may cause attacks of weakness, paleness and unconsciousness. The diagnosis must be made upon the absence of conditions causing hemorrhage. The frequent, fleeting unconsciousness of these patients, the presence of arteriosclerosis, and the history of previous attacks must influence the diagnosis. Here there may be a decided secondary anemia.

CEREBRAL ANEMIA.

Simple cerebral anemia (syncope) is accompanied by paleness, unconsciousness and feeble, rapid pulse. There is no hemorrhage, no disease which might cause faintness, but there is the presence of some mental shock—the sight of some unpleasant occurrence such as free blood. This latter, when it comes from the person's own body, may, *per se,* amount to nothing, but the sight of it may cause the patient to faint.

SHOCK.

Shock from a blow, especially upon the head or abdomen, is very often the cause of the symptoms of hemorrhage. Here care must be taken to exhaust every diagnostic means in order to exclude hemorrhage.

If time permit, an examination of the blood for hemoglobin and red cells will show a decrease of both in hemorrhage. There is no decrease of hemoglobin and red cells in any of the other conditions mentioned.

2. Anemia

The essential anemias which will be discussed are (*a*) chlorosis; (*b*) pernicious anemia.

(*a*) *Chlorosis*

Occurrence.—This disease occurs in young women between thirteen and eighteen years of age.

Symptoms.—The symptoms of the disease are cessation of the menstrual flow, weakness, lack of desire to do the usual work, dyspnea, palpitation of the heart, vertigo, tendency to syncope and pallor of the face, lips and conjunctivae. Occasionally the cheeks, instead of being pallid, are flushed or flush easily under excitement; but even in these cases there is marked pallor of the lips and conjunctivae.

Physical Signs.—On examination of the patient the heart is found to be of normal size or very slightly dilated, rapid in action, sometimes irregular, with a soft systolic murmur heard best at the base of the heart and in the vessels of the neck. Over the veins of the neck one can distinguish a constant to-and-fro murmur, the so-called bruit de diable. This bruit exists in extreme cases and will disappear when the patient lies down.

In eliciting both of these—the bruit and the systolic murmur in the vessels—great care must be taken that no pressure be made upon the vessel with the bell of the stethoscope. I say *bell of the stethoscope* advisedly, because use of the old single stethoscope is an extremely uncertain manner of eliciting these murmurs; at least it is difficult to *distinguish between a pressure murmur and a murmur which really exists in the vessel itself.*

The lungs are found to be normal; the abdomen is absolutely normal; there is no enlargement of the glands. The hemoglobin taken by a good instrument will show ten to thirty per cent of hemoglobin; the red corpuscles on the other hand will be diminished to between 3,500,000 to 2,000,000—rarely below 2,000,000. Therefore the color index is low; that is, each individual corpuscle has a much lower relative hemoglobin content than is normal. For instance, if the blood count is 2,000,000 and the hemoglobin count 10 per cent, the red cells are 40 per cent of normal and the hemoglobin 10 per cent of normal. The blood index will be one-quarter. The number of white cells is not changed from normal. A differential count of the blood will show that there may possibly be some change in the size and shape of the red corpuscles or they may take a stain badly, but there is no great degree of poikilocytosis as there is in pernicious anemia. There may occasionally be a nucleated red—a normoblast, that is, a normal sized red blood corpuscle with a nucleus.

Conditions to be Differentiated from Chlorosis

This disease must be differentiated from:

Pernicious anemia
Leukemia
Hodgkin's disease
Organic heart disease
Secondary anemia
Nephritis
Hyperthyroidism

PERNICIOUS ANEMIA.

Pernicious anemia rarely occurs in young individuals. The patient becomes gradually blanched and extremely weak; hemorrhages may occur from the mucous membrane or into the skin; there is much palpitation of the heart; there is much dyspnea; frequently the patellar reflexes are markedly decreased and the individual has many of the signs of locomotor ataxia. There is very marked blood change. The red cells sink to between one million and one-half and one million; the hemoglobin is low —often 10 to 15 per cent to 30 per cent; and a differential count shows much poikilocytosis and many nucleated reds. Here it will be observed that the color index is high. Megaloblasts, abnormally large nucleated red cells, are common. The relative number of small lymphocytes is frequently increased, whereas the total number is decreased.

LEUKEMIA.

Chlorosis is clinically distinguished from leukemia by the fact that there is no enlargement of the lymph glands; there is no enlargement of the spleen and the blood picture differs entirely. In chlorosis there is no change in the leukocytes; in leukemia there is the pathognomonic sign of leukocytosis and of a polymorphism of the leukocytes; that is, there may be a great increase of lymphocytes, myelocytes and eosinophils. Or the leukocytosis may exist in increase of the lymphocytes and there is always a relative decrease in the polymorphonuclear cells.

HODGKIN'S DISEASE.

Chlorosis is distinguished from Hodgkin's disease by the absence of marked enlargement of the lymphatic glands of the latter, by the lack of improvement under treatment, and by the condition of the blood. In Hodgkin's disease there is usually a slight leukocytosis, but there is no marked difference in the relation of the various kinds of leukocytes and the hemoglobin is not so markedly low as it is in chlorosis; the red cells are not materially changed.

ORGANIC HEART DISEASE.

A wrong diagnosis of cardiac disease is often made in chlorosis because of the cardiac murmurs which occur; this can only be overcome by careful physical examination. In organic disease of the heart, the size of the heart is often greater than normal and a murmur may occur at any of the orifices.

In chlorosis the heart is not enlarged to any extent; the murmurs are often soft, and practically all of them occur at the base of the heart. Many cases of simple chlorosis are diagnosed as heart disease, much to the detriment of the patient who is made to believe she is suffering from organic heart disease, whereas, as a matter of fact, there is simply a changed blood condition.

SECONDARY ANEMIA.

In looking over the journals of today, we do not see nearly the number of cases of chlorosis reported as formerly. This is due to the fact that better methods of examination have revealed many instances of incipient tuberculosis or syphilis, and of anemia due to small hemorrhages that were heretofore overlooked.

In order to make a diagnosis positive, one must be assured (1) that there is no tuberculosis of the glands or any other organ, (2) that the individual is not suffering from syphilis—and this latter may be ascertained by the use of the Wassermann reaction, and (3) that she is not suffering from a small unnoticed hemorrhage due to hemorrhoids, etc. In a word, every organ must be carefully scrutinized to ascertain whether or not it is the cause of the trouble. Malnutrition, or unhygienic work must also be excluded as a cause for the hemorrhage.

The blood findings in chlorosis are as above stated, whereas in hemorrhage or secondary anemia spoken of, the fall of the hemoglobin and of the corpuscles is more equable. For instance there may be 2,500,000 red cells and 50 per cent hemoglobin instead of 2,500,000 red cells and 10 per cent of hemoglobin as in chlorosis.

Nephritis.

In nephritis the individual is pale; there is edema; but the disproportion between the red cells and the hemoglobin is not as great as it is in chlorosis. Above all, albumin and tube casts are present in the urine and there is usually a high systolic blood pressure.

Hyperthyroidism.

As an essential part of its symptomatology hyperthyroidism has one or all of these symptoms: enlargement of the thyroid gland, tachycardia, exophthalmos and muscular tremor. These are absent or only temporary in chlorosis. There may be a secondary anemia.

(*b*) *Progressive Pernicious Anemia*

(*Primary or Essential Anemia*)

Cause.—So far as is at present known, this condition is the result of some impairment of the blood-making organs.

Symptoms.—It is characterized by the symptoms of simple exhaustion, inability to continue at one's ordinary duties, extreme paleness, loss of color, and blanching of the sclera and mucous membranes. Often there are gastro-intestinal crises with severe vomiting and diarrhea. There may be abolition of the reflexes and other marked symptoms of cord degeneration, such as ataxia, anesthesia, etc. It is characterized also by remissions not due to treatment (Plate 4, Fig. 3).

Blood Findings.—Examination of the blood shows a very marked reduction of the red cells, frequently to a million per cubic millimeter, and rarely above one million and one-half, with a high color index—that is, the red cells are reduced out of all proportion to the reduction of the hemoglobin. There are many normoblasts and megaloblasts. The red cells are changed in size and shape. A leukopenia is present, and the small lymphocytes often make up a large percentage of the white cells; there is often granular degeneration of the red cells.

Conditions to be Differentiated from Progressive Pernicious Anemia

This condition must be distinguished from:

Secondary anemia
Chlorosis
Cancer of the stomach
Leukemia
Hodgkin's disease
Locomotor ataxia and lateral sclerosis
Addison's disease
Splenic anemia.

SECONDARY ANEMIA.

Most secondary anemias can very readily be distinguished by the discovery of a primary disease and by the examination of the blood. As has been mentioned in speaking of general symptoms of anemia, the blood of a secondary anemia has a relatively equal reduction in hemoglobin and in red cells; this is in marked contrast to the remarkable reduction of red cells in pernicious anemia.

There are two conditions in which the anemia is secondary, however, in which it approaches pernicious essential anemia in every detail—that is, the anemia caused by the intestinal parasite, diborothocephalus, and in certain cases of syphilis. Here, as before said, the blood picture is in every way comparable to the blood picture of essential anemia, and these facts make it most important that the stools be examined and a Wassermann reaction be made in every case of severe anemia, in order to rule out the possible presence of this tapeworm or of syphilis.

The author has notes of one case of tertiary syphilis, in which the blood had every characteristic of progressive pernicious anemia.

CHLOROSIS.

Chlorosis is a disease of young girls, and is characterized by paleness of the skin and mucous membranes, by dyspnea, palpitation and weakness, by disturbance of the menstrual function. The age of the patient and the character of the blood should at once suggest the diagnosis. Pernicious anemia rarely occurs in young individuals. The condition of the blood differs entirely: there is little change in the red cells; they are frequently not reduced below three million; and the hemoglobin very markedly reduced. Treatment by hygiene and iron will cause an immediate amelioration.

Cancer of the Stomach.

The diagnosis of cancer of the stomach from pernicious anemia is frequently difficult to make, but in the former there is always the history of a long-standing indigestion; there is usually a change in the stomach contents characteristic of cancer of the stomach—loss of acidity, presence of lactic acid and presence of Oppler-Boas bacilli. Then, too, as the case progresses, a tumor usually is observed which marks the case as one of carcinoma. Here the use of the x-ray is of the greatest value because involvement of the stomach wall is usually accompanied by indentation of the shadow of the stomach wall. The blood in carcinoma does not have such persistent high color index, and there are fewer normoblasts and megaloblasts.

Leukemia.

In leukemia, as noted in the chapter on leukemia, anemia is one of the symptoms, but there is always an enlargement of glands, usually of the spleen, and the blood picture differs entirely, the blood picture of leukemia being that of increase of white cells and a moderate anemia.

Hodgkin's Disease.

Hodgkin's disease is characterized by enlarged lymphatic glands, especially of the cervical group, by a slight leukocytosis and by a rather moderate secondary anemia.

Locomotor ataxia and Lateral Sclerosis.

In some cases of pernicious anemia, vertigo and the gait of tabes dorsalis or of spastic paralysis are the symptoms which are most prominent. There is frequently loss of the patellar reflex. But the retention of the pupillary reflex, the history of the case together with the presence of the blood picture characteristic of pernicious anemia will make the diagnosis in favor of this latter disease, while a fairly normal blood with pupillary involvement and cord symptoms will favor locomotor ataxia.

While in diseases of the cord anemia may be one of the symptoms in certain cases, there is never the marked reduction of red cells nor other characteristics of pernicious anemia.

Addison's Disease.

Addison's disease is characterized by a progressive anemia, but with a peculiar pigmentation of the skin of dark brown character, which differs entirely from the lemon yellow color of pernicious anemia. Here the finding of tuberculosis in another portion of the body and extreme asthenia suggests the condition to be one of Addison's disease and not of pernicious anemia. The blood picture differs in all respects.

Splenic Anemia.

Splenic anemia is a secondary anemia with enlarged spleen and pigmentation of the skin. In the later stages of the condition there are symptoms which closely resemble those of cirrhosis of the liver; indeed, the symptoms are identical, there being a true cirrhosis of the liver.

The blood picture is that of a secondary anemia and is in no way comparable to that of pernicious anemia.

3. Leukemia

Symptoms.—This condition is characterized symptomatically by paleness of the mucous membrane and of the skin, by dyspnea, by palpitation of the heart, and at times by general anasarca.

Physical Signs.—Physical examination usually shows enlargement of the superficial lymph glands and enlargement of the spleen; the glandular enlargement may be so slight superficially that it cannot be detected or the spleen may be only slightly enlarged.

Diagnosis.—*Examination of the blood* without question will make the diagnosis positive. There is always an increase either in the number of the leukocytes or in the varieties of the leukocytes. Toward the fatal end of certain cases the number of leukocytes sinks to normal, but the abnormal character of the leukocytes still continues. This number reaches anywhere from 15,000 to 1,000,000 per cubic millimeter, not an unusual number being 200,000 to 300,000. The changes in the cells proper depend upon the character of the condition. In the so-called myelogenic form there is great diversity in the character of the leukocytes; there are polymorphonuclear cells present; there is an increase in the lymphocytes both large and small; also there is an increase in the eosinophils, in the mast cells and in most of the specimens. An entirely new cell is present, to wit: the myelocyte.

In addition to this change in the leukocytes, there is also a change in the red cells as shown by their size, color and form and also by the fact that many of them are nucleated.

In so-called lymphatic leukemia the change in the white cells consists of increase in the number of lymphocytes. In the acute form of leukemia these lymphocytes are either large or small, or the acute form may be myelogenous; while in chronic lymphatic leukemia, the increase is in the small lymphocytes.

PLATE V

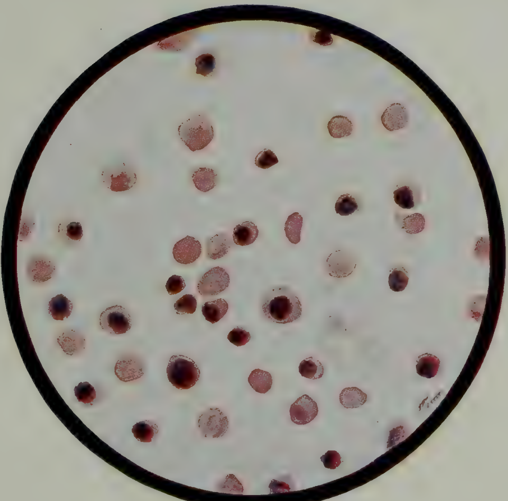


Fig. 1.

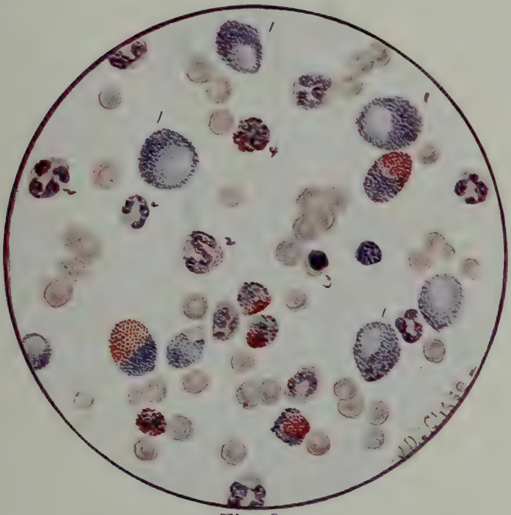


Fig. 2.

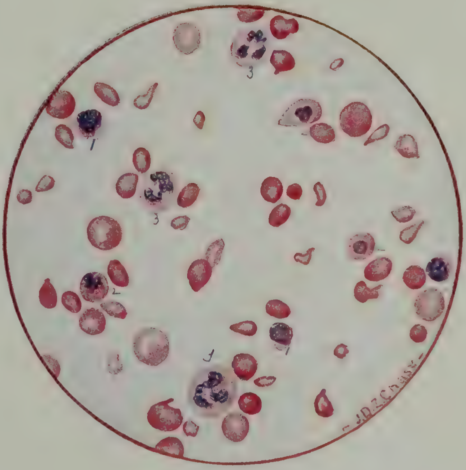


Fig. 3.

Fig. 1.—Blood of Chronic Lymphatic Leukemia. (Personal Observation.)
Fig. 2.—Blood of Myelogenous Leukemia. (Kindness of Dr. Orlando Petty.) 1, Myelocytes; 2, Polymorphonuclear Leukocytes; 3, Nucleated Red Cells; 4, Mast Cells.
Fig. 3.—Blood of Pernicious Anemia. (Author's Case.) 1, Small Lymphocyte; 2, Nucleated Red Corpuscles; 3, Polymorphonuclear Leukocytes.

Frequently there is fever of an entirely irregular character lasting a longer or shorter time accompanying the disease.

Conditions to be Differentiated from Leukemia

These conditions must be differentiated from:

Hodgkin's disease, pseudoleukemia
Pernicious anemia
Glandular tuberculosis
Septic infection
Splenic enlargement due to other causes.

HODGKIN'S DISEASE.

Hodgkin's disease is marked by a general glandular enlargement. It has a much more characteristic fever than has leukemia of the chronic form, the fever in Hodgkin's disease lasting for a number of weeks at a time and being intermittent in type.

There is some enlargement of the spleen. The differential diagnosis depends entirely upon the blood examination and the examination of an excised gland.

In Hodgkin's disease there is a moderate anemia; the white cells are frequently increased to 15,000 or 20,000. The blood platelets are increased, the neutrophils are increased. The characteristic blood picture described by Yates and Bunting will be found on page 485, under the heading, Hodgkin's Disease.

PERNICIOUS ANEMIA.

There may be some enlargement of the spleen in pernicious anemia but there is no glandular enlargement. The patient is of the lemon color which is quite characteristic.

The examination of the blood does not show the changes characteristic of leukemia; a picture peculiar to itself is seen—that is, a particularly marked reduction of the red cells, being usually below 1,500,000 to the cubic millimeter; the hemoglobin is relatively high (though actually low) so that the color index is high. In addition there is marked poikilocytosis and nucleation of the red cells. The white cells in pernicious anemia are frequently reduced in number; the small lymphocytes are very often in the majority, often as high as 80 per cent.

GLANDULAR TUBERCULOSIS.

In tuberculosis of the lymph glands there is some enlargement of the glands as in Hodgkin's disease and in leukemia; there may be fever which is very irregular in type; examination of the glands will show the presence of tubercle bacilli, but the blood picture is not at all characteristic, and in no way resembles either leukemia or Hodgkin's disease.

SEPTIC INFECTION.

The condition of leukemia might possibly be mistaken for a septic infection as there may be an increase often as high as 60,000 white cells in sepsis, but the increase occurs altogether in the polymorphonuclear cells; there are no changes in the character of the red blood cells.

Acute Leukemia

Symptoms.—The acute form of leukemia is one which comes under the family physician's eye. It is characterized by a continued fever, often ranging from 103°-104° F., with very little diurnal variation, and frequently with the appearance of a NEPHRITIS—albumin and casts in the urine, at times with diarrhea. Hemorrhage is common; there is leukocytosis.

A case reported by Taylor, Jopson and Fussell (Philadelphia Med. J., Jan. 7, 1899) illustrates this well.

Diagnosis.—Acute leukemia may be mistaken for almost any acute infection, but particularly for TYPHOID FEVER.

The differential diagnosis can only be made by examination of the blood; the characteristics of leukemia will be found. This is another evidence of the value of routine blood examinations and of a complete blood examination including a differential count.

Some rare cases are on record, notably one of the writer's, in which acute leukemia was mistaken for DIPHTHERIA. In this case the condition began with an acute inflammation of the tonsil overlaid with a dark gangrenous membrane.

In a case of acute leukemia the examination of a culture from the throat shows the absence of diphtheria bacilli, and examination of the blood will show the characteristics of leukemia.

4. Hodgkin's Disease

Hodgkin's disease occurs in both males and females and is characterized by general glandular enlargement. This enlargement, however, is often marked at first and also appears to the greatest degree in the glands of the neck, great masses appearing in both sides of the neck.

In advanced cases the substernal glands enlarge and cause pressure, the case then resembling one of mediastinal tumor. There is some enlargement of the spleen; there is absence of signs of tuberculosis in other portions of the body than the glands, marked pallor and fever, the latter of continued type coming on in paroxysms, each paroxysm lasting from one to three weeks. The glands do not suppurate.

The following blood picture, according to Yates and Bunting, is typical of the disease:

There are two types of Hodgkin's disease, an early and a late, showing a constant characteristic increase in the number of platelets with abnormally larger forms and either a relative or absolute increase in the so-called transitional cells.

In the early type the leukocytes are usually less than 10,000; very early there is a moderate increase in the basophils, and when the disease is well established, the eosinophils are slightly increased. The polymorphonuclear neutrophils remain within the usual limit, the lymphocytes at or slightly above normal.

The late type shows a leukocytosis which may reach 100,000, the neutrophils are relatively increased to a percentage of 75 to 92, and the lymphocytes to 5 per cent, frequently even less. The transitionals—the only other cells of large number—are usually above 8 per cent, unless the leukocytosis is very high, when they may be relatively fewer, but still exceed the lymphocytes in number.

Conditions to be Differentiated from Hodgkin's Disease

Leukemia
Tubercular adenitis
Syphilitic adenitis
Mediastinal tumor.

LEUKEMIA.

Leukemia is at once differentiated by examination of the blood which shows increase of the white cells, usually to an enormous quantity—frequently above 100,000 leukocytes to the cm.—and with a polymorphous change, often the presence of myelocytes and eosinophils in unusual numbers.

TUBERCULAR ADENITIS.

Tubercular adenitis is not so easily differentiated, although in the blood of such cases there is usually a lymphocytosis and not the typical blood picture described above. The glands are prone to break down and suppurate; this is not the case in Hodgkin's disease. There is a tuberculin reaction in the skin.

SYPHILITIC ADENITIS.

In syphilitic adenitis there is no change in the white blood cells. The Wassermann reaction is present here, and is negative in Hodgkin's disease.

MEDIASTINAL TUMOR.

Mediastinal tumor might be mistaken for Hodgkin's disease in cases in which there is a general sarcomatosis and where there is also a mass under the sternum. As stated above, occasionally the mediastinal glands so enlarge in Hodgkin's disease that a veritable mediastinal tumor, giving rise to pressure symptoms, results. In malignant disease there is usually a primary source of the condition discoverable. The superficial glands are usually the first affected in Hodgkin's disease. Examination of any superficial enlarged glands, in cases of mediastinal tumor, will show the characteristic sarcomatous type and not the type belonging to Hodgkin's disease.

5. Purpura

Characteristic Features.—This is a condition characterized by hemorrhages from the mucous membranes, bleeding of the gums, subcutaneous hemorrhages, hematuria and sometimes bleeding into the serous membranes. The purpuric spots can be differentiated from an erythema by the fact that they do not disappear when pressure is made upon them. Purpura, in reality, is a symptom and not a separate disease.

It varies in intensity from small subcutaneous hemorrhages, entirely without symptoms, to a severe condition causing grave anemia and dangerous hemorrhages from the lungs, kidneys, bowels or stomach. Its cause is probably made active by a lowered vitality, by exposure, alcoholism, by many drugs and by almost any infection. It is frequently present in such varied conditions as nephritis, pernicious anemia, scarlet fever, syphilis, meningitis, scurvy and leukemia.

Various Forms.—Different forms are given different names, *peliosis rheumatica* being applied to petechial spots occurring chiefly on the legs and accompanied by severe pains in the joints. Apparently it has nothing in common with rheumatism except the painful joints.

Simple purpura is characterized by slight subcutaneous hemorrhages with a feeling of uneasiness and digestive disturbances, such as diarrhea, etc.

Purpura hemorrhagica is the most severe form of purpura, characterized by severe subcutaneous hemorrhages and bleeding from the kidneys, lungs, stomach and bowels, and often hemorrhages into the serous cavities.

The writer has notes of a case of this character, accompanied by a severe but recovering hemiplegia. Between these two extremes there are many graduations.

Then there are the attacks of *purpura in the course of infectious diseases*, such as scarlet fever, typhus fever, typhoid fever, and indeed in any infection of severity.

Alcoholism and arteriosclerosis of severe grades are often accompanied by subcutaneous hemorrhages, entirely symptomless as far as the hemorrhage itself is concerned.

Certain poisonous substances, such as snake venom, and drugs, such as copaiba and quinin, when absorbed *are frequently accompanied by quite severe hemorrhages.* The writer has now under treatment a woman of 80 years, in whom one-half grain of quinin in two doses caused a severe erythema, many of the spots being hemorrhagic.

Hemorrhages of the newborn are characterized by rapid anemia, malena, subcutaneous hemorrhages and often jaundice (hematogenous).

Scurvy, which writers all class as a fault in metabolism, seems to the writer to be merely a form of *severe purpura,* in which there is distinct symptomatology; hardship, faulty food and exposure, are perhaps known to be the exciting causes.

Conditions to be Differentiated from Purpura

Purpura must be distinguished from:

Scurvy
Hemophilia
Acute leukemia.

SCURVY:

Scurvy, as above stated, has distinct symptomatology—weakness, pallor, bleeding gums, enlargement of the glands, induration of the legs which is said to be characteristic, and in very advanced cases, necrosis of the bones.

A distinct history of deprivation, of absence of food substances found in fresh vegetables, of great hardship, and in the scurvy of infants, the elements present in mother's fresh milk are necessary in order to make a diagnosis of scurvy.

HEMOPHILIA.

In hemophilia heredity is always the important factor. A person in apparent health may bleed from a scratch, a drawn tooth, or any slight cut. It occurs practically always in males, and is transmitted through the females of a bleeder family.

ACUTE LEUKEMIA.

Acute leukemia, in which severe hemorrhages often occur, is at once diagnosed by the increase of leukocytes in the blood. There is no characteristic change in the blood of purpura.

6. Hemophilia

Characteristic Features.—Hemophilia is a disease characterized by hemorrhages from unimportant lesions of the surface of the body and by subcutaneous hemorrhages; it is further characterized by the fact that it occurs only in males, but is transmitted through females. Usually the bleeding first appears at an early age; it may be so severe as to threaten life, even though the lesion be very small.

There may be severe ecchymoses in various portions of the body, there may be severe epistaxis, or the extraction of a tooth may cause uncontrollable bleeding—even a prick or a scratch may cause severe bleeding. Hemorrhages also occur from the mouth and into the various joints.

Conditions to be Differentiated from Hemophilia

This condition may be mistaken for:

Purpura
Scurvy.

PURPURA.

Purpura, as is stated above, often has petechial spots upon the surface of the body, accompanied at times by severe hematuria or hematuresis as initial symptoms. Sometimes the petechiae are the only symptoms. Frequently there is leukocytosis and often fever, the individual being without exception in a lowered state of vigor owing to some infection, whereas in hemophilia the bleeding occurs in the midst of perfect health without any other symptom than bleeding. In purpura there is no history of bleeding in other members of the family as there is in hemophilia.

SCURVY.

Scurvy might be mistaken for hemophilia for much the same reasons as is purpura, but here there is the history of exposure, insufficient and improper food; also there is marked bleeding of the gums without any previous traumatism. The bones in scurvy are often the seat of subperiosteal hemorrhages. These do not occur in hemophilia.

Perhaps the most important diagnostic point is the exposure to improper food and hardship. The feeding of infants with artificial food is a frequent source of scurvy. It has no relation to hemophilia. The facts that the inheritance of hemophilia is through the mother in almost all cases, and that the condition of hemophilia occurs only in males, are extremely important.

It must be remembered that in neither purpura nor scurvy is there an actual hemorrhagic diathesis, while in hemophilia there is this transmitted tendency of a diathesis.

The writer has seen two cases of persistent purpura which bled on the least disturbance, but both of these persons were alcoholics; in neither was there any heredity.

7. Erythremia

(Polycythemia, Osler's Disease)

Characteristic Features.—This condition is characterized by remarkable increase of red cells in the peripheral blood, together with an increase of the hemoglobin, enlargement of the spleen, and frequently cyanosis. The increase in the blood cells often amounts to 13,000,000 per cubic millimeter. The hemoglobin may reach 130 to 150 per cent. There is usually dyspnea and high blood pressure and frequently nephritis.

Conditions to be Differentiated from Erythremia

The condition may be mistaken for:

Congenital heart disease
Cyanosis due to emphysema
Pseudocyanosis due to ingestion of coal tar products or to intestinal absorption.

CONGENITAL HEART DISEASE.

Congenital heart disease may resemble polycythemia in that there is marked cyanosis of the fingers, of the feet and face; but examination of the heart will show that there is a distinct organic lesion of that organ and a general examination will show clubbed fingers, marked dyspnea and lack of development of the patient.

Examination of the blood will show an increase of red cells but will not show the very *marked* increase of erythrocytes and hemoglobin which are present in erythremia; also there is no enlargement of the spleen.

CYANOSIS DUE TO EMPHYSEMA.

Emphysema may be accompanied by a cyanosis, but the diagnosis can be made certain by a careful examination of the chest. The examination of the blood and chest will of course reveal the picture characteristic of emphysema. There is no enlargement of the spleen in emphysema. Examination of the blood in these cases will not show the blood picture of erythremia.

INGESTION OF COAL TAR PRODUCTS.

Individuals who are accustomed to taking large quantities of coal tar products develop a discoloration of the skin which simulates a cyanosis.

Here the appearance of the whole body is of a slate color, as contrasted with a true cyanosis; the mucous membranes also partake of this discoloration in hemoglobinemic cyanosis.

This condition differs from polycythemia in the following particulars: the blood count is normal, there is no enlargement of the spleen, and the heart's action is quite normal.

There exists also an enterogenous erythremia which occurs as the result of some intestinal disturbance. The diagnosis must be made by the presence of an intestinal toxemia and the absence of the blood count of erythremia. One must be certain of the absence of drug taking in these latter cases.

8. Hemoglobinemic Cyanosis

This symptom may be caused by intestinal stasis (enterogenous cyanosis) or it may result from the ingestion of certain drugs, such as potassium chlorate, carbon monoxid, sulphonal, trional, and coal tar products. The coal tar products which are particularly culpable are acetanilid and acetphenetidin. Here the whole body is of a slate color which is not due to diseases of the heart or lungs.

Conditions to be Differentiated from Hemoglobinemic Cyanosis

It can be and frequently is mistaken for:

Diseases of the heart
Diseases of the lungs
Erythremia
Argyria.

DISEASES OF THE HEART AND LUNGS.

These diseases can be differentiated by the fact that there is present marked variation in the function of these organs, which is not present in coal tar poisoning or in enterogenous cyanosis. An attempt to take the hemoglobin with the ordinary instruments is futile in hemoglobinemic cyanosis; the spectroscope must be used to discover the presence of methemoglobin and sulphemoglobin.

ERYTHREMIA.

Erythremia (polycythemia) might be mistaken for this disease but for the following characteristic symptoms: examination of the blood shows very marked increase of red cells and of hemoglobin and increase in the size of the spleen. These are absent in cases of hemoglobinemic cyanosis.

ARGYRIA.

Argyria differs from hemoglobinemic cyanosis in the history of the case. On careful questioning it will be discovered that in a case of argyria the patient has been taking nitrate of silver for one reason or another, while the cyanosis cases, almost without exception, give a history of drug taking for headache, or they are the subjects of gastro-intestinal disturbances. Then, too, in argyria, a discoloration of the skin cannot be made to disappear by pressure. The spectroscope must be used to study the blood coloring matter.

The differentiation of *enterogenous cyanosis* and cyanosis due to drug taking is extremely difficult. The former condition is due either to the presence of methemoglobin or sulphemoglobin, and only examination of the blood by an expert spectroscopist will decide the diagnosis. To make the diagnosis of the enterogenous cyanosis from the drug form, it is absolutely necessary to first exclude the taking of drugs.

Section X

Diseases of the Ductless Glands

A. Diseases of the Suprarenal Bodies

Addison's Disease

Cause.—This condition is due to a change in the adrenal glands.

Characteristic Symptoms.—It is characterized by extreme asthenia and pigmentation of the skin, varying in color from gray or light yellow to dark. The mucous membranes are often affected. Occasionally the pigmentation affects the mucous membranes. Gastro-intestinal symptoms characterized by vomiting and diarrhea are common.

The symptoms first noticed are usually pigmentation of the skin, followed by gastro-intestinal symptoms, and finally by marked asthenia, with low blood pressure.

Diagnosis.—The use of adrenal extract in cases of pigmentation with low pressure is said by Grünbaum (Practitioner, Aug., 1907) to be of the greatest value in diagnosticating the disease from other forms of pigmentation. The blood pressure in Addison's disease is abnormally low. The use of three grains of adrenal preparation, representing three to five grains of desiccated gland, for three days, will, according to Grünbaum, raise the blood pressure. If the rise is more than 10 per cent, it is excellent evidence that the case is one of true Addison's disease, as compared with pigmentation due to other causes.

The presence of tuberculosis in some other portion of the body, with the symptoms of asthenia, low blood pressure and gastro-intestinal symptoms, is good evidence that the adrenals are involved. It must not be forgotten, however, that asthenia and gastro-intestinal symptoms are frequent symptoms of tuberculosis in other portions of the body than the adrenal glands. The pigmentation in such cases, therefore, would be the deciding symptom, together with the sign of raised blood pressure by the use of desiccated gland.

Conditions to be Differentiated from Addison's Disease

Other conditions which cause pigmentation of the skin are:

Pernicious anemia
Exophthalmic goiter
Malignant disease
Jaundice
Pigmentation due to vermin
Argyria
Arsenical poisoning
Vitiligo
Pregnancy
Bronzed diabetes
Malarial fever
Arthritis deformans
Neurasthenia with gastro-intestinal symptoms
Interstitial nephritis
Pellagra
Diseases of the ovaries.

Of this motley group of conditions with pigmentation, pernicious anemia, exophthalmic goiter, malignant disease and neurasthenia with gastro-intestinal symptoms will give the greatest trouble in differentiation.

PERNICIOUS ANEMIA.

Pernicious anemia is, as a rule, accompanied by a lemon-colored skin. This might be mistaken for the pigmentation of Addison's disease, particularly when affecting some of the southern races, as the Italians and Greeks. There is the same pearly whiteness of the sclera and the same extreme asthenia, but the discoloration in cases of anemia does not affect the mucous membranes. In pernicious anemia there is frequently—indeed it is the rule—remission of symptoms, during which the patient returns to symptomatic good health; this is not the case in Addison's disease. Examination of the blood will make certain the diagnosis between the two conditions.

The characteristic blood findings in pernicious anemia—extreme reduction of red cells, low hemoglobin but a high color index, poikilocytosis, anisocytosis, polychromatophilia and nucleated red cells—are in marked contrast to the relatively mild secondary anemia of Addison's disease.

EXOPHTHALMIC GOITER.

In cases of exophthalmic goiter pigmentation of the skin frequently appears about the exposed surface, but the mucous membranes are not pigmented. There is a marked tachycardia, which differs from the rapid heart of Addison's disease in that the cardiac dullness is more diffuse; there is much more violent cardiac impulse; there is enlargement of the thyroid gland, with a murmur over the gland; there is muscular tremor and exophthalmos. None of these are present in Addison's disease.

Malignant Disease.

Malignant disease, especially of the ovaries and sarcoma of the ovary, is frequently accompanied by pigmentation, but here, though the patient is weak and distressed, one does not find the very marked asthenia seen in Addison's disease. Then, too, the mucous membranes are not pigmented, and a mass is frequently present, which can be more or less accurately diagnosed.

Jaundice.

Jaundice may be distinguished from the pigmentation of Addison's disease by the fact that the yellow discoloration of jaundice always affects the sclera. The sclera in Addison's disease is pearly white. It is also to be distinguished by the rare occurrence of extreme weakness and low blood pressure which accompany Addison's disease. In jaundice there is a history of gall-stones, obstruction from a mass, acute gastric duodenitis, none of which precede Addison's disease. The urine is bile-stained—which is not the case in Addison's disease.

Pigmentation Due to Vermin.

Pigmentation due to vermin—vagrant's pigmentation—is accompanied by scratch marks. There is no asthenia unless the patient is ill from some other disease, nor is there pigmentation of the mucous membranes.

Argyria.

Argyria is accompanied by a slate-colored appearance of the skin; there is the history of the consumption of nitrate of silver for some time, but there are no signs of involvement of the general health.

Arsenical Poisoning.

Arsenical poisoning may occur in individuals working with arsenic, in those who have the arsenic habit, and in individuals who are taking the drug in therapeutic doses; but here again there are no evidences of failure of general health except in those who are taking the drug for some constitutional disease, and in those instances the symptoms of the original disease can be recognized.

Vitiligo.

Vitiligo, with its dark pigmentation interspersed with spots of total loss of pigment, and immediately adjoining them, should be recognized by its usual chronicity and by the fact that there is no general disease discoverable.

PLATE VI

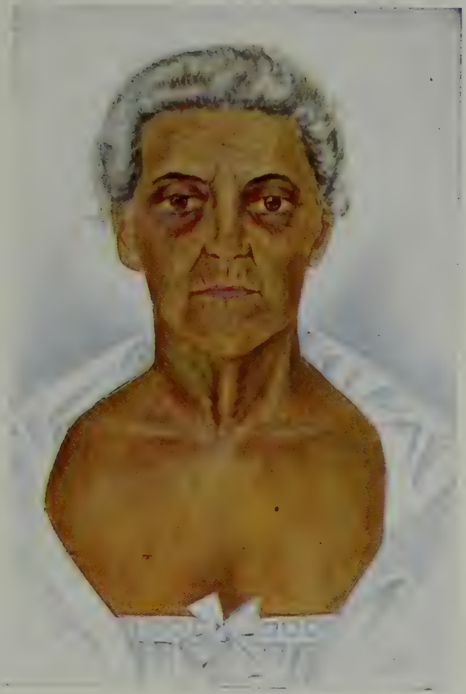


Fig. 1.

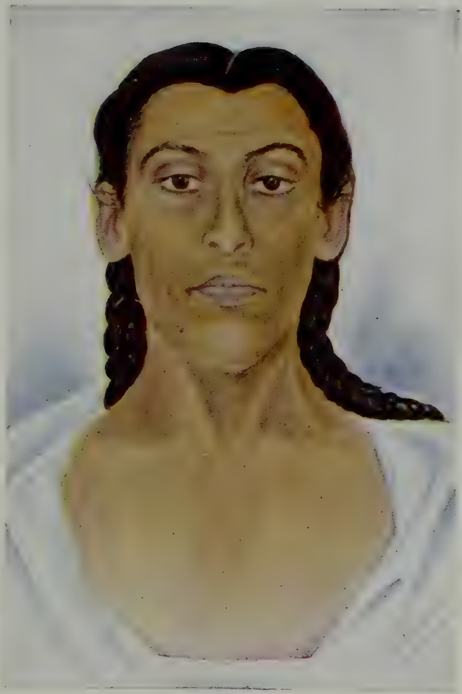


Fig. 2.

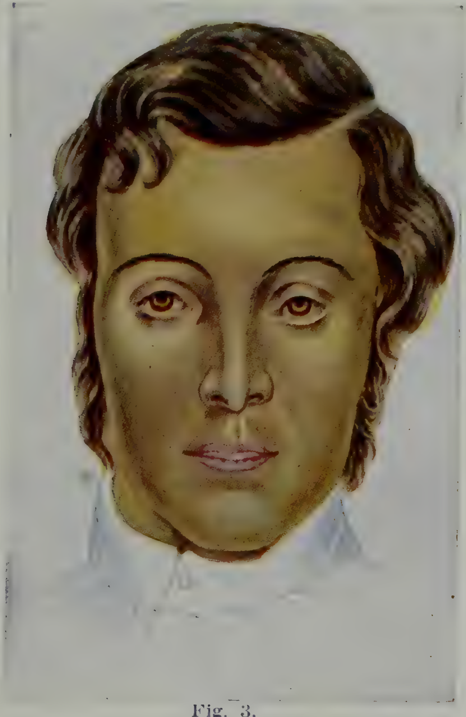


Fig. 3.

Fig. 1.—Jaundice in a Case of Cancer of the Pancreas. (Author's Case.)
Fig. 2.—Pernicious Anemia in a Greek. (Author's Case.)
Fig. 3.—Addison's Disease. (From Addison's Original Publication, Library of College of Physicians, Philadelphia, Pa.)

Pregnancy.

Pregnancy, which is sometimes accompanied by pigmentation, is so evident in history and the characteristic mass that it is almost impossible to mistake it for Addison's disease.

Bronzed Diabetes.

Bronzed diabetes is differentiated by means of the urine examination and by the enlarged liver. Sugar does not appear in the urine of individuals with Addison's disease; neither does asthenia occur in diabetes to the degree that it does in Addison's disease.

Malarial Fever.

Chronic malarial poisoning is often accompanied by a severe pigmentation, but the presence of plasmodia in the blood, enlarged spleen and the history of acute attacks of malarial fever will influence the diagnosis.

Arthritis deformans.

Arthritis deformans is always accompanied by characteristic changes in the joints which are unmistakable, and which never occur in Addison's disease.

Neurasthenia with Gastro-intestinal Symptoms.

Neurasthenic individuals may occasionally be pigmented from various causes. When these persons are badly affected by their condition one may be justified in admitting the possibility of so severe and fatal a disease as Addison's.

Careful examination will alone make the diagnosis. The neurasthenic individual is always fearful of some impending evil; the rapidity of the heart is much less when the patient is at rest; there is no cardiac dilatation; there is no organic lesion to be found anywhere.

Interstitial Nephritis.

In interstitial nephritis, in certain rare instances, patchy pigmentation is also noted, but the hypertrophy of the heart, the elevated blood pressure and the urine of low specific gravity, containing tube casts, will enable one to promptly make an accurate diagnosis in favor of interstitial nephritis.

Pellagra.

This condition lacks the marked asthenia of Addison's disease, though the gastro-intestinal symptoms, together with the pigmentation, might be mistaken for Addison's disease. The appearance of the rash of pellagra on the hands and face and the occurrence of the condition in individuals of all ages will help to make the diagnosis.

DIFFERENTIAL TABLE

Disease	Asthenia	Pigmentation	Circulatory Changes	Blood Changes	Urine	Tumor	Lungs	Gastro-intestinal Tract	Symptoms and Physical Signs
Addison's Disease	Marked	Dark brown to light Mucous membranes affected	Rapid heart; low blood pressure	Secondary anemia	No change	None	Often signs of tuberculosis	Vomiting and diarrhea	Great weakness; rapid heart; slight loss of flesh. Pigmentations; low blood pressure
Exophthalmic Goiter	Not marked	Occasional about face	Rapid dilated heart	None	No change	Thyroid gland enlarged and pulsating	No change	Diarrheal attacks	Tremor; exophthalmos; rapid heart
Neurasthenia	Not constant	Slight at certain times	Changeable, sometimes rapid, peripheral pulsation	Slight anemia	No change	Palpitating aorta	No change	No change	No change
Vermin Pigmentation	None	Dirt and scratch marks	None	None	No change	None	No change	No change	Itching; history of finding of lice
Vitiligo	None	Dark and white spots interspersed	None	None	No change	None	No change	No change	No change
Malarial Fever	Weak after paroxysm	About exposed portions	None except in attack	Malarial organisms	No change	Enlarged spleen	No change	No change. Vomiting in attack	Chills, fever, malarial district
Jaundice	None	Yellow uniform mucous membranes	None	None	Bile in urine	None	No change	Indigestion	Possible abdominal pain and indigestion
Argyria	None	Slate color, involving lips	No change	No change	No change	None	No change	No change	History of taking silver preparations
Arsenical Poisoning	None	Brown	No change	No change unless from primary disease	No change	None	No change	No change	Symptoms of disease for which arsenic given. Arsenic eating

DIFFERENTIAL TABLE—*Continued*

Disease	Asthenia	Pigmentation	Circulatory Changes	Blood Changes	Urine	Tumor	Lungs	Gastro-intestinal Tract	Symptoms and Physical Signs
Pernicious Anemia....	Weak and breathless	On face; mucous membranes not affected	Weak heart; hemic murmurs	Low blood count; red cells below normal 2,000,000; nucleated reds	No change	None	No change	Diarrhea in spells	Weakness; paleness; remission of attack
Diseases of Ovaries...	None	On face	None	None	No change	Probably of ovary	No change	No change	Tenderness in pelvis; pelvic or abdominal mass
Malignant Disease...	Weakness	About face: not on mucous membranes	Weak heart	Secondary anemia	No change	In various parts	No change	Indigestion, if gastro-intestinal tract involved	Weakness Tumor Indigestion
Pregnancy...........	None	About face and nipples	No change	No change	Possible albuminuria	Abdominal mass. Uterus enlarged	No change	Early vomiting	Cessation of menstruation
Bronzed Diabetes.....	None	Face and hands	Weak heart	None	Sugar	Enlarged liver	No change	No change	Sugar in urine. Enlarged liver. Pigmentation of skin
Arthritis deformans...	None	Occurs about face and joints	None	None	No change	Enlarged painful joints	No change	No change	Painful joints; swollen joints; stiffness
Interstitial Nephritis	Weak	Occasionally occurs	Enlarged heart; high blood pressure	Secondary anemia	Albuminuria. Low sp. gr. Granular tube casts	None	No change	Indigestion	Dyspnea Polyuria Weakness
Pellagra	None	Erythema on face and hands	None	None	None	None	No change	Diarrhea; vomiting	Int. disturbance. Erythema, mental changes

B. Diseases of the Thymus Gland

The thymus gland is larger at birth than at any other time; it atrophies gradually with the growth of the individual. This accounts for the fact that practically all the diseases of this gland are diseases of early life.

1. Status lymphaticus

Morbid Anatomy.—This is a condition of general lymphatic enlargement over the entire body, associated with an unusually enlarged thymus gland. The child grows pale, becomes waxy in appearance, and peculiarly liable to sudden enlargements of the lymphatic glands upon the slightest infection. The thymus gland takes part in this enlargement. In anesthesia and under certain conditions—the nature of which is unknown—the child is seized with sudden respiratory difficulty, followed by death, which occurs almost instantly. In these states of thymic death the thymus gland is always found enlarged. In practically all these cases there is an antecedent history of tracheal compression, with attacks of asthma, more or less sudden.

Conditions to be Differentiated from Status lymphaticus

The condition of status lymphaticus is difficult to distinguish from a general lymphatic enlargement due to tuberculosis, syphilis and rickets—any one of which may be associated with enlargement of the thymus gland.

SYPHILIS.

This condition can be differentiated by the history of skin eruptions of syphilis at birth or very shortly after, and especially and certainly by the presence of a positive Wassermann reaction.

RICKETS.

Rickets has the characteristic square head and chest, the open fontanels, the enlarged epiphyses, bowed legs and arms—none of which are present in so-called status lymphaticus.

GENERAL LYMPHATIC ENLARGEMENT DUE TO TUBERCULOSIS.

General enlargement of the lymph glands due to tuberculosis is more difficult of diagnostication; however, the enlargement under the sternum, as seen by the x-ray, is differently placed from that seen in lymphatic enlargement.

2. Enlarged Thymus

This is the most important of all the conditions connected with the thymus gland.

Occurrence.—It usually occurs soon after birth, but may occur at any time from then on until the thymus disappears entirely.

Symptoms.—The symptoms are difficult inspiration and expiration, often amounting to an actual stridor. The stridor is loudest while the child cries; it does not disappear during sleep, but occasionally disappears suddenly for a few minutes without apparent cause, to reappear again. The inspiratory and expiratory acts are both prolonged, the former being more marked. There are signs of obstruction in the breathing; these are shown by the retraction, both in the supersternal notch and epigastrium, and by the presence of loud tracheal sounds. There is dullness of greater or less extent under the upper piece of the sternum; often the dullness extends beyond the line of the upper piece of the sternum. Cyanosis and edema of the lungs occur, and intracranial pressure is manifest. The x-ray will show a shadow under the upper part of the sternum. There may be a mere stridor, there may be attacks of acute symptoms due to compression, or there may be sudden death. The severity of the symptoms depends upon the degree of compression on the trachea; intubation does not relieve the stridor; there is no change in the voice.

Conditions to be Differentiated from Enlarged Thymus

The condition must be differentiated from:

Diphtheritic croup
Catarrhal laryngitis
Laryngismus stridulus
Compression of the trachea by peribronchial glands
Compression on the trachea by new growths
Adenoids
Malformation of the larynx
Foreign body in the air passages.

Diphtheritic Croup—Catarrhal Laryngitis.

These are both acute conditions; neither is congenital, and in neither is there a history of long-continued difficult breathing. The patient is hoarse; examination of the larynx shows redness of the cords or an exudate.

In diphtheria there is the history of hoarseness a few hours before the onset of severe symptoms. An important point to be remembered is that in diphtheria, almost without exception, an exudate can be found on the pharynx or on the tonsils. Diphtheria bacilli may be discovered by culture of the throat exudate.

In acute catarrhal laryngitis there is the history of a slight cold. Suddenly—usually during the night—the individual is seized with a hoarse cough, croupy in character, and with symptoms of suffocation.

Enlarged thymus differs from these in the fact that, almost without exception, there is a history of more or less stridor for days and weeks preceding a possible acute attack, though in the rarer cases there may be a sudden attack, at times ending fatally—occurring in a child apparently in perfect health. A difference in the physical signs is also noted: there is dullness under the sternum in thymus, which is not evident in catarrhal laryngitis or diphtheria, and an x-ray picture, taken if time permits, will show a shadow under the sternum in enlarged thymus which is absent in laryngitis.

Laryngismus stridulus.

Laryngismus stridulus is more difficult to differentiate; but here the individual is usually rickety; there is no dullness under the sternum, and an x-ray will not show the picture of thymus enlargement. Then, too, in listening over the trachea, a point low down will show the seat of the greatest compression by auscultation in thymic enlargement, while in laryngismus stridulus it will be higher up over the larynx.

Compression of Trachea by Peribronchial Glands.

A tumor composed of enlarged lymphatic glands may easily be mistaken for thymic enlargement. However, the dullness in thymic enlargement is noted anteriorally, whereas the dullness in bronchial enlargement is located posteriorly; in addition, the stridor is both inspiratory and expiratory in thymus, whereas in glandular enlargement it is expiratory.

Compression on the Trachea by New Growths.

Much more difficulty will be experienced if there is a mediastinal tumor of malignant origin. Here only careful, repeated examinations with the x-ray with percussion and continued observation of the patient will discover the condition to be really a malignant one and not the result of thymic enlargement.

Adenoids.

In adenoids the obstruction can be located in the pharynx by ocular and digital examination. The sound disappears when the nares are held closed.

MALFORMATION OF THE LARYNX.

Congenital stridor due to malformation of the larynx may be diagnosed by ocular examination or digital examination of the larynx. The larynx is not affected in thymic disease.

FOREIGN BODY IN AIR PASSAGES.

A foreign body lodged in the larynx or bronchus may simulate pressure from an enlarged thymus gland, but in this condition there is usually the history of the inspiration of a foreign body. The symptoms come on in the midst of good health; there is no dullness under the sternum; there are often attacks of violent coughing, and a Röntgen examination will frequently show a shadow produced by the foreign substance.

C. Diseases of the Thyroid Gland

Diseases of the thyroid gland may be classified as follows:

Inflammation of the Thyroid Gland
New Growths
Diseases Due to Atrophy of the Gland Substance
Diseases Due to Deficiency of the Thyroid Secretion—Hypothyroidism
Diseases Due to Excess of the Thyroid Secretion.

1. Thyroiditis

Symptoms.—In thyroiditis there are the symptoms of an acute infection, together with enlargement, swelling and tenderness in the gland itself.

This enlargement gives rise to pain and often to signs of pressure. Hoarseness may be present, due to involvement of the recurrent laryngeal nerve (Dock).

Conditions to be Differentiated from Thyroiditis

This may be mistaken for:

Enlargement due to simple congestion
Tuberculosis
Actinomycosis
Syphilis
Other tumors of the neck
Carcinoma
Acute goiter.

The presence of a specific infectious disease, a focus of pus, and the fact that the individual has an acute infectious condition will serve to distinguish thyroiditis from any of these.

Enlargement Due to Simple Congestion.

Simple congestion of the thyroid gland is not accompanied by signs of general infection; it may be the result of sexual excitement, especially in the female.

Tuberculosis.

Tuberculosis of the thyroid gland may be either miliary or nodular. The miliary infection occurs in practically all cases of general miliary tuberculosis; nodular tuberculosis closely resembles malignant disease. If there is tuberculosis of the lungs or other organs, a nodular growth may be taken for a malignant one. If the nodule caseates or softens and tubercle bacilli can be demonstrated, of course a diagnosis can be made. According to Dock, the condition is so difficult of differentiation from malignant cases that it is advisable to remove the gland in most cases.

Actinomycosis.

Actinomycosis can be distinguished by enlargement of the gland and demonstration of the organism peculiar to the condition. This latter can only be done by examining the material coming from a softened nodule or by aspiration of material from the nodule.

Syphilis.

Luetic enlargement of the thyroid gland, according to Dock (quoting from Engel-Reimers), occurs in about one-half the cases of recent infections. It is soft and painless, and disappears very slowly under specific treatment. It may be recognized by the presence of a Wassermann reaction and the absence of symptoms of other causes of enlargement.

Echinococcosis.

This enlargement is rare. It can be differentiated from other enlargement by the presence of a cystic, either multilocular or simple, tumor and demonstration of hooklets in the contained liquid.

Malignant Tumors.

These may be either sarcomata or carcinomata. They cause enlargement of the gland, the enlargement taking place rather suddenly, either in a normal gland or in a goiter. Primary growths in other portions of the body will make the diagnosis certain.

2. Goiter—Struma

Chronic simple goiter enlargement of the gland may involve one or both lateral lobes or it may involve only the middle lobe. This disease is characterized by a soft elastic growth, varying in size from a simple observable enlargement to a huge mass the size of a head. There are no general symptoms except those caused by pressure of the gland. These may be dyspnea, dysphasia and signs of obstructed breathing, vertigo due to pressure upon the vessels of the neck, as also palpitation of the heart in certain cases (Fig. 96).

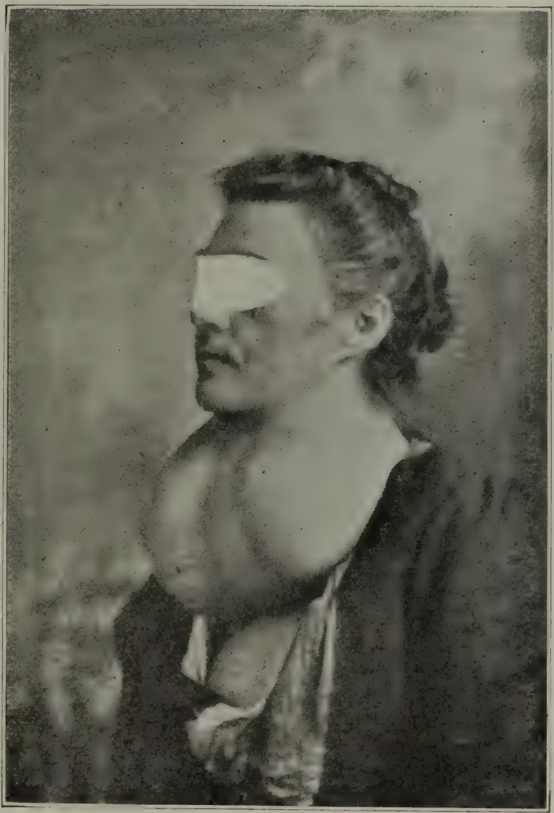


Fig. 96.—Large Cystic Goiter Measuring 31 Inches in Circumference, Including Neck. (From Mayo's Clinic.)

Conditions to be Differentiated from Goiter

The conditions with which this disease may be confounded are:

Carcinoma or malignant degeneration
Enlargement of the cervical lymph glands
Other tumors of the thyroid gland
Exophthalmic goiter.

The goiterous enlargement of the gland itself can practically always be distinguished from a growth outside the gland, except when of a very great size, by its softness, its elasticity and its upward and downward movement in the act of swallowing.

CARCINOMA.

In carcinoma the gland is hard and painful, the patient loses weight, and there may be symptoms of involvement of the general system.

ENLARGEMENT OF THE CERVICAL LYMPH GLANDS.

Enlargement of the cervical lymph glands is usually nodular; it does not move with the act of swallowing; it is not in the median line, but rather on one or the other side.

EXOPHTHALMIC GOITER.

A simple colloid degeneration of the thyroid gland often merges into one of oversecretion or hyperthyroidism. In the condition of simple enlargement, however, there is no exophthalmos, no tremor, no tachycardia and no thrill or murmur, which are present in exophthalmic goiter.

For differentiation of symptoms of other enlargements, see page 502.

3. Exophthalmic Goiter—Hyperthyroidism

(*Basedow's Disease, Graves's Disease*)

Cause.—The condition caused by the hypersecretion of the thyroid gland is known as exophthalmic goiter or hyperthyroidism.

Characteristic Features.—The fully developed disease is characterized by an enlarged pulsating thyroid gland, exophthalmos, tachycardia and muscular tremors. The enlargement of the gland may vary from the size of a lemon to that of a large fist.

This *enlargement of the gland is characterized* by its soft character and a very marked pulsation *in the gland itself*, by a palpable thrill and an audible systolic murmur or a double murmur over the gland. Both the pulsation over the gland and the murmur may be closely simulated by the murmur transmitted from the carotid arteries, where these arteries pulsate either on account of disease of the heart, or of anemia, neurasthenia, or diseases of the artery itself.

The exophthalmos is characterized by marked protrusion of the eyeballs, varying from a mere prominence of the eye to a condition in which the eye becomes so prominent that the lids do not cover the ball when the lids are closed. True exophthalmos may be known by the so-called Stellwag's sign, in which the palpebral angle is decidedly greater than normal; it is also characterized by von Graefe's sign. This sign is demonstrated in the following manner: the patient is asked to look upward, following with the eye an object slowly lowered from above the point of vision to a point below the field of vision. When this is done it will be found that the eyelid lags behind the eyeball; in the normal individual the eyelids follow the eyeball.

The circulatory condition is characterized by extremely rapid heart action, whether the patient is in a recumbent or in an upright position, whether excited or quiet, by marked pulsation of the peripheral vessels, the carotids, the subclavians, and of the vessels of the legs and arms. Occasionally there is arhythmia.

The muscular tremor is characterized by fine tremors—principally in mild cases—in the arms and hands. It can best be developed by asking the patient to hold his arms horizontally away from his body. It may be so severe as to involve the entire body, shaking the head and extremities.

In addition to these cardinal symptoms in a well-developed case, when the patient looks upward the forehead does not wrinkle. In these cases there is a continuous hum heard over the protruding eye when the bell of the stethoscope is placed over this organ (Riesman). A marked tendency to attacks of vomiting and diarrhea is seen; also a tendency to hyperemia. Fever is of common occurrence. The patient becomes extremely dyspneic, and complains of palpitation of the heart, weakness and dyspnea, and is often emaciated.

The superficial appearance of a well-developed case of exophthalmic goiter is characterized by so much prominence of the eyes that the term "frog face" is often applied to it.

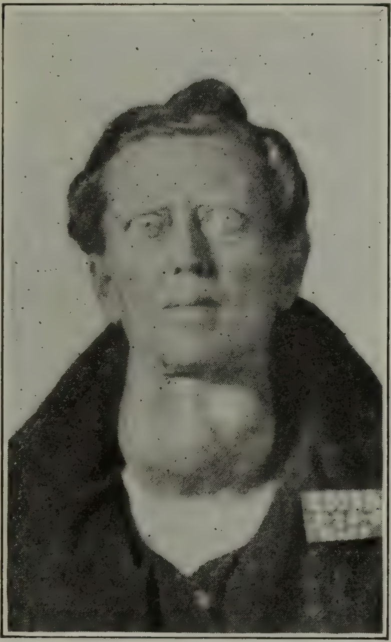


Fig. 97.—Exophthalmic Goiter. (From Mayo's Clinic.)

Various Grades of Hyperthyroidism.—There are all grades of this hyperthyroidism, varying from the typical case which I have mentioned to a simple nervousness characterized by an attack of cardiac irritability, muscular tremor, with only slight exophthalmos, and slight, if any, enlargement of the thyroid gland.

Conditions to be Differentiated from Exophthalmic Goiter

The disease is to be differentiated from:

Neurasthenia
Simple enlargement of the thyroid gland
Exophthalmos due to local causes
Cardiac disease, notably aortic regurgitation
Interstitial nephritis.

NEURASTHENIA.

Neurasthenia may closely simulate the disease. There may be marked muscular tremors, but as a rule the tremor is coarse—not fine as it is in hyperthyroidism; there may be marked pulsation of the peripheral vessels as marked as in any case of exophthalmic goiter. The pulsation is particularly noticeable in the abdominal aorta, but in these cases of neurasthenia there is no exophthalmos, there is no enlargement of the thyroid gland, or if, by a coincidence, there should be an enlargement of the gland, such as a simple goiter, there is no murmur over the gland nor is there a thrill, unless it be transmitted. Great care must be taken in these cases of pulsation in the vessels of the neck due to neurasthenia not to mistake the transmitted murmur which is heard over the gland for a murmur originating in the gland itself. This distinction may be easily made by shutting off the pulsation in the vessels by pressure below, or by lifting the gland from the underlying vessels. There may be tachycardia in cases of neurasthenia, but the tachycardia, as mentioned before, is not persistent and is much quieter when the patient is asleep than awake. There is rarely the to-and-fro murmur over the base of the heart, which is frequently present in exophthalmic goiter. There is no exophthalmos in the case, and if the eye does seem prominent it may be differentiated from exophthalmos by the absence of the von Graefe and Stellwag signs.

SIMPLE ENLARGEMENT OF THE THYROID GLAND.

Simple goiter may be differentiated by the entire absence of general symptoms, such as tachycardia, muscular tremors, exophthalmos and lack of pulsation and murmur in the gland.

EXOPHTHALMOS DUE TO LOCAL CAUSES.

Exophthalmos due to local causes is likely to be unilateral, although it may at times be bilateral, in which instances there is an entire absence of the general symptoms above described.

CARDIAC DISEASE.

Cardiac disease, notably aortic regurgitation, has marked peripheral pulsation and marked tachycardia as one of its symptoms, but there are the positive signs of organic heart disease, such as enlargement of the heart and the presence of endocardial murmurs. There is an absence of exophthalmos or thyroid enlargement and of murmurs over the thyroid gland, but here again there may be a transmitted murmur over the thyroid gland, and this must be distinguished by the methods above stated.

INTERSTITIAL NEPHRITIS.

As pointed out by Barker, this disease often has exophthalmos as one of its symptoms, but there is not the tremor, cardiac palpitation and goiter, which are present in Graves's disease, and there are the urinary changes.

4. Myxedema—Hypothyroidism

Deficient secretion of the thyroid gland is characteristic in the disease known as myxedema.

This disease may be *congenital,* when the individual is known as a cretin, or it may be *acquired,* either by the result of removal of the gland or disease of the gland. In the latter condition it affects adult females rather more frequently than males.

Congenital Myxedema.—A cretin gives evidence of deficiency of the thyroid gland early in life. The little patient, instead of being bright, taking notice, attempting to sit up or walk, is dull, listless and expressionless, the tongue protrudes, saliva drips from the wide open mouth, there is drooling; the patient does not sit up, does not attempt to walk, the skin becomes thick and dry, and the hair fails to grow. The cretin who has grown to the age of three or four years has heretofore been frequently mistaken for an idiot, but since thyroid extract has been administered in these cases the senseless, dull, unintelligent individual is changed into a bright, rational being, who crawls, sits up, walks, and quickly grows in height.

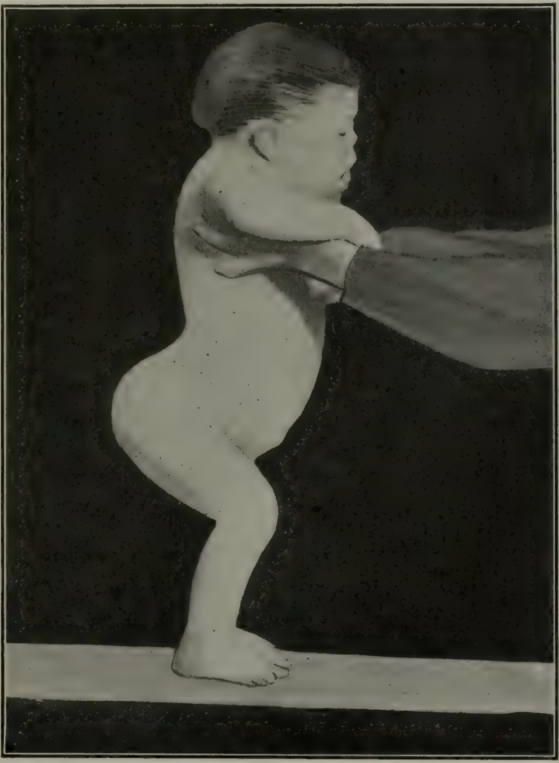


Fig. 98.—Sporadic Cretinism. (From Am. Textbook of Children's Diseases, W. B. Saunders Co.)

Adult Myxedema.—Myxedema in the adult is characterized at first by listlessness. A heretofore cheerful and bright person who has been attending to the ordinary duties of life suddenly desires to be inactive and to sleep on the slightest pretext. The face grows expressionless, the skin becomes thick and dry and does not pit on pressure, the hair becomes thin and dry, he experiences the sensations of cold, has tinnitus aurium and often deafness. The sexual instincts are abolished, the face is bloated, there is no smile, saliva often dribbles from the lips of the individual, and the patient has the appearance of one with some chronic, overpowering disease of the brain. There is anemia; nucleated red cells are not uncommon in the blood.

Treatment.—Incomplete forms of this condition sometimes occur where many of the symptoms of a fully developed case are lacking. Immediate improvement of these cases will occur if desiccated thyroid gland extract is administered.

The administration of thyroid extract to these individuals is astounding. Following the continuous administration of thyroid extract for 48, 72 or 96 hours, the patient loses weight, the skin becomes dry, the patient becomes bright and quite normal in appearance within two or three weeks.

Conditions to be Differentiated from Myxedema

This disease must be distinguished from:

Idiocy (in the infant)
Organic brain disease
Nephritis
Disease of the heart
Pernicious anemia.

IDIOCY (IN THE INFANT).

A child with diseased brain or underdeveloped brain does not have thick, dry, harsh skin like the cretin; it does not have the pads of fat above the clavicle, nor does it have the spadelike hands. The administration of the thyroid extract does not have any effect in these cases.

ORGANIC BRAIN DISEASE IN THE ADULT.

Disease of the brain in adults lacks the characteristic thick dry skin with thin dry hair; it lacks the cardiac symptoms of myxedema and the blood changes in myxedema; and, again, the administration of thyroid extract has no effect.

NEPHRITIS.

In chronic parenchymatous nephritis the patient is anemic, the skin is edematous, the urine of a very high or very low specific gravity, containing albumin and tube casts. There is dyspnea and hypertrophy of the heart. The condition of the kidneys will decide the diagnosis.

DISEASE OF THE HEART.

In the disease of the heart, the organic change of the heart wall or the valves and the absence of typical symptoms of myxedema influence the diagnosis.

Pernicious Anemia.

Pernicious anemia is sometimes simulated by the fact that the blood picture of myxedema is often that of extreme anemia. However, there are various features in which they differ: the mentality of a patient with pernicious anemia is not affected; any swelling which occurs is a true edema; weakness, failure of strength, and extreme breathlessness are the first and chief symptoms of progressive anemia as contrasted with a dull intellect, inability to think, and dry thick skin of an individual with myxedema.

The administration of thyroid extract has no effect in cases of pernicious anemia.

D. Diseases of the Parathyroid Glands

1. Tetany

Etiology.—It has been fairly well established that this condition is due to a disease or to removal of the parathyroid glands, these small glands having to do with the calcium metabolism in the body.

Occurrence.—Tetany is quite common in young children and in pregnant women. It also occurs in certain cases of infectious diseases, such as typhoid fever, scarlet fever, measles, cholera and in certain intoxications such as uremia and lead poisoning. It is also found in certain cases of dilatation of the stomach.

Symptoms.—The chief symptoms are chronic, continuous or intermittent spasms of the extremities, of a tonic character; it is also characterized by attacks of laryngismus stridulus.

The hand may be tightly closed, the thumbs drawn in and the hand flexed upon the wrist. The toes become extended, as is the foot at the ankle.

An attack may often be brought on by firm pressure over the great nerves of the arm or leg—the so-called Trousseau's sign. Tapping over the facial nerve will bring on a spasm of the face—Chvostek's sign.

Laryngismus stridulus is characterized by long drawn inspiration and expiration similar to croup. There is no fever and no sore throat. The condition may last months or weeks.

Conditions to be Differentiated from Tetany

Tetany may be mistaken for:

Croup
Organic brain disease
Convulsions other than tetany
Tetanus.

CROUP.

Croup is practically always due either to a simple acute laryngitis or it is the result of diphtheritic laryngitis. There is fever, exudate in the throat or simple redness of the fauces. If the case is diphtheritic, diphtheria bacilli can be demonstrated. It is acute and short in duration.

ORGANIC BRAIN DISEASE.

Organic brain lesions bring about contractions of one or all of the extremities. There is frequently spasmodic contracture of the extremities, but in these cases there is likely to be permanent disability. The attack cannot be precipitated by pressure over the nerve trunks or vessels nor can Chvostek's sign be developed, as in tetany.

CONVULSIONS OTHER THAN TETANY.

Reflex convulsions are usually general; they are clonic in character; there is unconsciousness for a greater or less time; there is often fever, of which the convulsion is one of the results, or the convulsion may result from reflex causes, such as indiscretions of diet, intestinal worms, etc.

TETANUS.

Tetanus is an infectious disease, the result of a traumatism and infection by the tetanus bacillus. In these cases there is the history of a traumatism; the convulsions are general. Frequently the disease is rapidly fatal.

E. Diseases of the Spleen

1. Splenic Anemia

Symptoms and Physical Signs.—Splenic anemia—primary splenomegaly with anemia—is a chronic condition, with the symptoms of chronically enlarged spleen, progressive anemia and leukopenia. There is a tendency to hemorrhages from the various mucous membranes, especially of the stomach, and sometimes in the skin. The skin has a marked tendency to become pigmented. There is frequently continued fever.

It is understood that this division may embrace several conditions, which will be separated later. In the late stages there arises a typical portal cirrhosis of the liver (Banti's disease), which resembles closely Laennec's cirrhosis or portal cirrhosis.

The symptoms of the anemia are those usually met with in that condition—breathlessness, vertigo and cardiac palpitation, with a low blood count.

The spleen is often enormous; in one case now under observation the spleen reaches to the pelvis. The liver is sometimes enlarged in the later stage—the Banti form; it is atrophic. Ascites may occur when the case resembles that of ordinary cirrhosis of the liver, but the spleen is always much larger than in ordinary cirrhosis.

The anemia is of the secondary type. The blood is not characteristic. The red cells may be almost normal in number or may fall to 2,000,000—rarely below. The low counts are found usually in those suffering from hemorrhage. There is not the same degree of change in the red cells as there is in pernicious anemia. The hemoglobin is usually lower in proportion than the red cells—it averaged 45 per cent in Osler's cases. There is usually a leukopenia.

Conditions to be Differentiated from Splenic Anemia

This condition must be separated from the various other states in the symptomatology, of which splenomegaly forms a part:

Leukemia
Pseudoleukemia
Malarial cachexia
Pernicious anemia
Syphilis
Cirrhosis of the liver
Hemorrhage
Tumor of left kidney
Amyloid spleen
Gaucher's splenomegaly
Splenic neoplasm.

LEUKEMIA.

This condition is at once distinguished on examination of the blood. In leukemia, as a very general rule, there is a leukocytosis. The leukocytosis is made up of various types of cells, namely, myelocytes, lymphocytes, polymorphonuclears and eosinophils. If the type is lymphatic there is an increase in the lymphocytes. There is less pigmentation in leukemia than in splenic anemia. In splenic anemia there is a very marked tendency to pigmentation, which is in contrast to the marked sallow paleness of leukemia.

PSEUDOLEUKEMIA.

In pseudoleukemia—Hodgkin's disease—there is fever of quite irregular type with a chronic enlargement of the lymphatic glands; but there is none of the peculiar pigmentation common in splenic anemia. The patient, on the other hand, becomes extremely white; there is not the marked enlargement of the spleen which is common in splenic anemia.

MALARIAL CACHEXIA.

Malarial cachexia may have splenomegaly as one of its symptoms, which must be differentiated from splenic anemia by the history, by residence in malarial districts, and by the presence of malarial organisms in the blood. There often is a secondary anemia in malarial cachexia, which must not be mistaken for the condition in splenic anemia. The fever which accompanies splenic anemia may, to a certain extent, correspond to the fever which occurs in malaria, but with careful blood examination in cases of malaria the malarial organism can always be discovered.

PERNICIOUS ANEMIA.

In pernicious anemia there is not the enlarged spleen of splenic anemia. The pigmentation which occurs is of the lemon yellow jaundice-like appearance, in contrast to the dark pigmentation of splenic anemia, and the blood picture differs entirely; the color index is higher, there is more destruction of the red cells. Then, too, the signs of cirrhosis of the liver which occur in the late stage of splenic anemia never occur in any cases of pernicious anemia.

SYPHILIS.

In its tertiary stage syphilis gives rise to anemia and an enlarged spleen. The history of syphilis and the presence of a Wassermann reaction will help make the diagnosis positive.

CIRRHOSIS OF THE LIVER.

In the later stages of splenic anemia—so-called Banti's disease—a cirrhosis of the liver occurs which exactly simulates Laennec's cirrhosis (portal cirrhosis). Laennec's cirrhosis, however, does *not* have the history of long-standing splenomegaly with a gradually increasing anemia. The first symptoms of Laennec's cirrhosis, on the other hand, are those of indigestion, with a possible hematemesis, a beginning ascites. The anemia is not so extreme. There is usually a history of alcoholism, which is not the case in splenic anemia.

HEMORRHAGE.

The gastric hemorrhage which occurs might be mistaken for a gastric hemorrhage from any other cause.

In gastric ulcer there will be the history of epigastric pain in relation to food common to that disease, and there is not the enlarged spleen which is present is splenic anemia, though cases of splenic anemia are on record where hematemesis is an early symptom.

Mitral stenosis is characterized by the cardiac findings in that condition. Cancer of the stomach can scarcely be confounded because of the marked systemic disturbance in cancer.

In bronzed diabetes there is always sugar in the urine.

TUMOR OF THE LEFT KIDNEY.

This tumor is usually lower; the edge is more rounded, the characteristic notch is not found; the dullness does not extend as far up under the ribs as that due to enlarged spleen. Usually there is some abnormality of the urine.

AMYLOID SPLEEN.

This degeneration of the spleen is a symptom of amyloid degeneration. There is the history of long-standing suppuration, or tuberculosis or of syphilis. Usually there is albuminuria.

GAUCHER'S SPLENOMEGALY.

Gaucher's splenomegaly was formerly thought to be a form of splenic anemia, but it has a special pathology. There are areas in the spleen which have characteristic large round or oval cells.

It begins in early life, and often appears in more than one member of the family. The liver enlarges; there is no gastric hemorrhage; ascites does not occur.

2. Movable Spleen

A movable spleen is differentiated from other tumors of the abdominal cavity by its size, its shape and by its position. A movable spleen may be felt in any position of the abdomen—the writer has seen one incarcerated in the pelvis. It has been felt as far over as the right side of the abdomen. It is recognized by the notch, which is characteristic of the spleen, by the ability to move the organ back into a normal splenic position, and by the entire absence of splenic dullness in the normal position.

Conditions to be Differentiated from Movable Spleen

Movable spleen may be mistaken for any movable abdominal mass, but particularly for the kidney.

MOVABLE KIDNEY.

A movable kidney is more likely to give signs of obstruction of the ureter—the so-called Dietl's crisis; there are no such symptoms and signs in a movable spleen.

The edge of the kidney is rounded and not sharp, it does not extend upward as does that of a splenic tumor. When the organ is incarcerated it gives rise to all of the signs of local abdominal inflammation. Unless the spleen can be outlined in these cases there is no possibility of making a diagnosis except by the absence of symptoms characteristic of a new growth.

PYLORIC TUMOR.

A pyloric tumor may be very movable and may be felt far over toward the splenic region, but the tumor does not have the outline of the spleen and there are signs of gastric disturbance which are not present in a movable spleen.

MOVABLE LIVER.

A movable liver is on the right side of the body. Its large size and position at once enable one to make a diagnosis.

GALL-BLADDER.

The writer has seen a gall-bladder dilate and with such an elongated neck that it could be moved to any portion of the abdomen. However, it was cystic and did not have the shape of the spleen; there were no symptoms.

3. Ruptured Spleen

Cause.—Ruptured spleen occurs as the result of traumatism.

Occurrence.—A few cases of spontaneous rupture are reported by Lewis A. Conner and William A. Downes (Am. J. Med. Sci., March, 1914). This same rupture is found in tropical malarial fevers, in a number of cases of typhus fever and relapsing fever. It was also reported in twelve cases of typhoid fever by Melchoir.

Physical Signs.—The signs of rupture of the spleen are those of shock and hemorrhage. Pain is often the first symptom. Local tenderness occurs if the rupture is a complication of one of the fevers, and tentative diagnosis can be made if local signs of inflammatory trouble occur in the region of the spleen followed by signs of internal hemorrhage.

Differentiation.—The condition might be mistaken for HEMORRHAGE FROM AN ULCER IN THE COURSE OF TYPHOID FEVER OR FOR PERFORATION IN TYPHOID FEVER. If the hemorrhage is due to ulcer, blood will appear in the bowel movements; a perforated intestinal ulcer will be followed by signs of general peritonitis and disappearance of liver dullness unless an operation is immediately done.

F. Diseases of Pituitary Gland

The pituitary gland is essential to normal life.

It controls the growth of the skeletal tissues, bone, and cartilage. It has to do with the development of fat, and holds some relationship with other ductless glands, especially the thyroid, adrenals, and sexual glands. It is materially influenced during pregnancy; changes induced by pregnancy are said to be so prominent that from them it can be averred that the subject has been pregnant. Its secretion, like that of the suprarenal glands, inhibits the pancreatic secretion, and may therefore have some casual relation to glycosuria. Its secretion increases blood pressure, and would seem to coöperate with the adrenals and to antagonize the thyroid.

Cushing in his book, "The Pituitary Body and Its Disorders, 1913," reviews all the literature and gives the most recent exposition of this most interesting gland. He classifies all the disorders of the pituitary body under the head of dyspituitarism. This author divides cases based upon experimental evidence and the observation of fifty clinical cases as follows:

> "Group I. Cases of dyspituitarism in which not only the signs indicating distortion of neighboring structures but also the symptoms betraying the effects of altered glandular activity are outspoken.
>
> Group II. Cases in which the neighborhood manifestations are pronounced but the glandular symptoms are absent or inconspicuous.
>
> Group III. Cases in which neighborhood manifestations are absent or inconspicuous though glandular symptoms are pronounced and unmistakable.
>
> Group IV. Cases in which obvious distant cerebral lesions are accompanied by symptomatic indications of secondary involvement.
>
> Group V. Cases with a polyglandular syndrome in which the functional disturbances on the part of the hypophysis are merely one, and not a predominant feature of a general involvement of the ductless glands."

One of the marked results of Cushing's studies is the establishment of the fact that cases which are affected by the hyperactivity of the pituitary gland show first the result of this overactivity in either gigantism or acromegaly, and later develop symptoms indicative of hypoactivity of the gland, these becoming mixed cases.

He divides the symptomatology of disturbance of the gland as follows:

> "(1) Neighborhood symptoms, (2) general pressure manifestations, (3) the secretory or glandular symptoms proper, and (4) the polyglandular manifestations."

Headaches are common, severe, and persistent.

Deformity of the sella turcica can be ascertained in certain cases by the x-ray. Atrophy of the optic nerves and disturbance of the fields of vision are among the common results of certain tumors.

Epistaxsis and marked discharge of mucus from the pharynx often supposed to be due to sinusitis are often due to pituitary disease.

Acromegaly and gigantism are the result of hyperplasia in the gland, the former after adolescence, the latter when young.

Launois and Roy say "gigantism is acromegaly in individuals whose epiphyseal cartilages are not ossified, whatever may be their age." In other words, both acromegaly and gigantism are the same condition brought about by overactivity of the gland occurring at different ages. In Cushing's words:

> "The disease is short, is the expression of a functional instability of the pars anterior, doubtless brought about by some underlying biochemical disturbance which leads to the elaboration of a perverted or exaggerated secretion containing a hormone that accelerates skeletal growth (the long bones if epiphyseal union is incomplete, of the sacral parts if epiphyseal ossification has taken place). Since the functional disturbance is probably a fluctuating one, with periods of increase

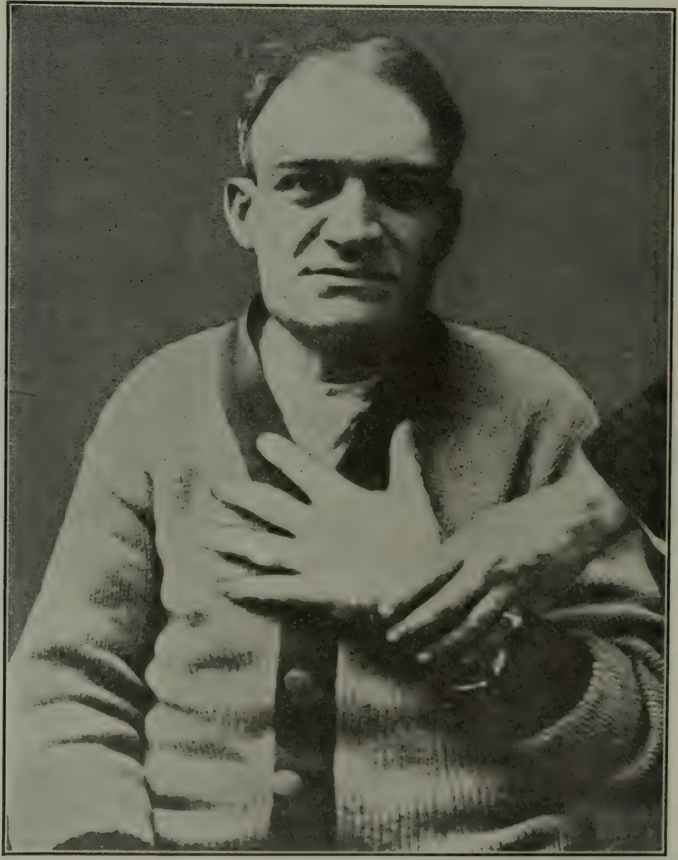


Fig. 99.—Hand in Acromegaly. From Case in Protestant Episcopal Hospital. (Kindness of Dr. Hooker.)

and remission, epiphyseal ossification may occur during a period of quiescence in the disorder. A subsequent recrudescence with resumption of the perverted functional activity will then serve to superimpose acromegalic manifestations on primary gigantism. Acromegaly, in other words, cannot precede gigantism, but may occur as gigantism which has become acromegalized.

"The most striking features are the enlarged bones, especially those of the hands and feet, the appearance of the former being well characterized as spadelike, while the fingers and nails are broad. The legs and arms, on the other hand, are not elongated early, but late in the disease; and the forearms and legs may increase in circumference, while the ends of long bones like the femurs, are often prominent. The scapula, clavicles, sternum and the ends of the ribs are also sometimes involved. The proper use of the hands is not interfered with. The head and face are enlarged, the spaces between the teeth are increased, while the neck appears short and the inferior maxilla may project beyond the upper, and the lower lip protrude in consequence. The ears are unduly prominent, while the cartilages of the nose, eyelids and larynx are enlarged and thickened as is also sometimes the tongue. The spinal column may be involved and there may be kyphosis. The muscles, on the other hand, are sometimes atrophied and the genitalia are unusually developed.

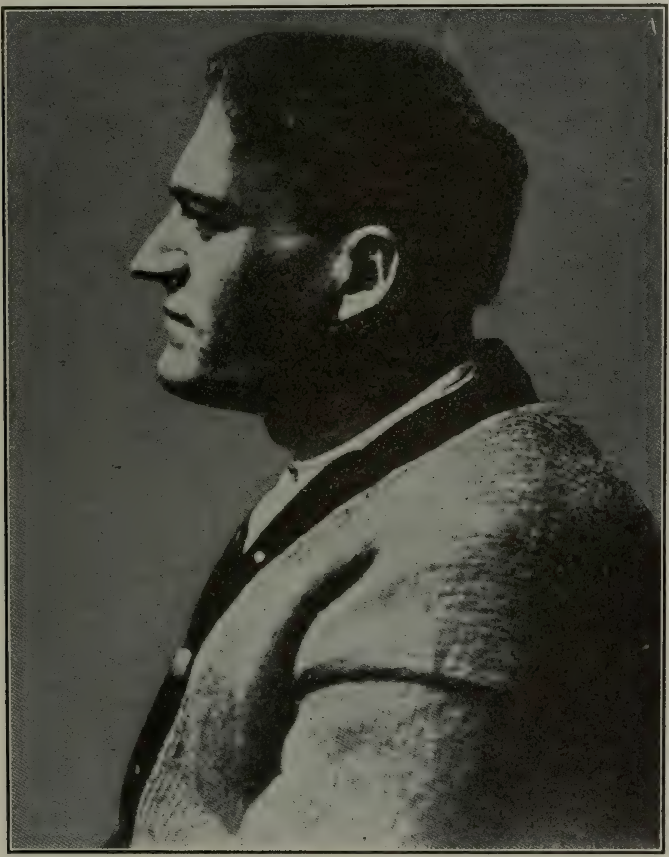


Fig. 100.—Dr. Hooker's Case of Acromegaly, Showing Prominent Nose. (From Episcopal Hospital Reports.)

The skin, though coarse and exhibiting a tendency to perspire, is not thickened as in myxedema.

"Among other symptoms are mental dullness, a sense of fatigue, and quite severe pain in the head and extremities, alteration of voice due to changes in the tongue and larynx, and possibly to paresis of the vocal cords; impairment of special sense of taste, smell and hearing; blindness, due to optic atrophy; thirst, shortness of breath, asthmatic attacks, palpitation, and even hypertrophy of the heart. In a number of cases bitemporal hemianopsia has been observed, which was due to pressure on the chiasm by the enlarged pituitary body. There are menstrual derangement and early cessation of the menses in women. The alterations in the thyroid have been alluded to, and an area of dullness over the manubrium is ascribed by Erb to persistence of the thymus.

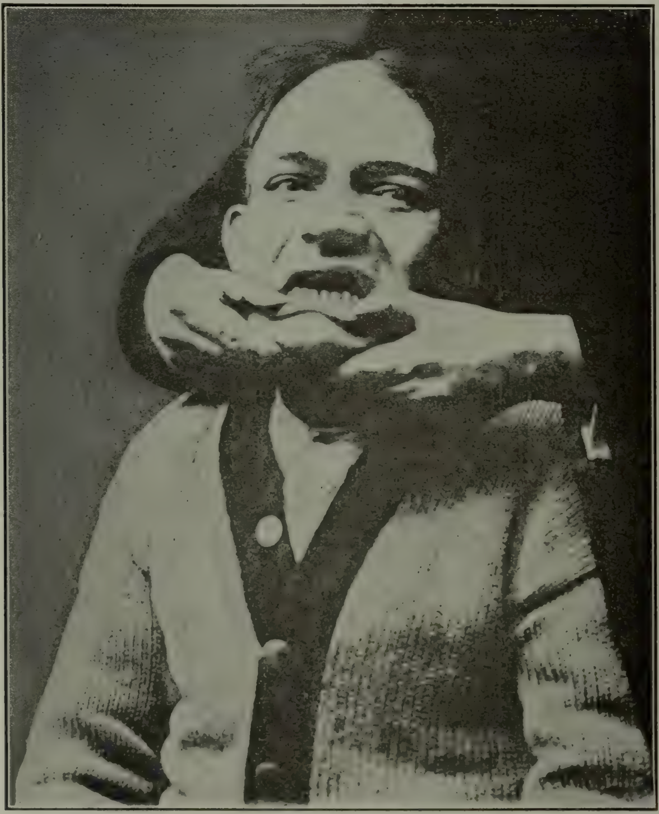


Fig. 101.—Showing Spacing of Teeth in Acromegaly. (Kindness of Dr. Hooker.)

"Hypopituitarism causes skeletal undergrowth which occurs when glandular insufficiency begins before the full stature is attained. When lack of activity begins before adolescence the skeleton has the feminine type."

Adiposity is common in cases where there is a lack of activity of the gland. He believes:

"We have attributed this particular symptom complex of adiposity, high sugar tolerance, subnormal temperature, slowed pulse, asthenia and drowsiness to a secretory deficiency of the posterior lobe; and a further argument in favor of this view is the reverse condition—namely, the emaciation, spontaneous glycosuria with hyperglycemia, and the slightly elevated temperature—which follows posterior lobe administration.

"Polyuria and polydypsia have followed sella decompression. Blood pressure is low in cases of hypopituitarism.

"Drowsiness and mental inactivity are also symptoms which occur. Certain symptoms—pigmentation of the skin, asthenia, low blood pressure, and to these may be added hypoglycemia—often occur in hypopituitarism, which greatly resembles Addison's disease."

Conditions to be Differentiated from Diseases of the Pituitary Gland

This condition can be confused with:

OSTEITIS DEFORMANS, but the bones in this condition are thick, the face is triangular, the cranial bones are "bossed," the teeth are unduly spaced, there is bowing of the legs, arms and clavicles.

Hypopituitarism may be mistaken for OBESITY FROM OTHER CAUSES AND INFANTILISM FROM OTHER CAUSES. The carbohydrate tolerance, however, in obesity and infantilism, not pituitary in origin, is not increased.

In the Lorain type of infantilism the individual is simply a small individual apparently normal in all other respects.

The fat cretin or myxedematous individual has thick skin, scant hair, marked mental determination, and is helped by the administration of thyroid gland preparations.

An achondroplastic has characteristic bone changes, bowed legs, pug nose, and may be unusually bright mentally. There is no change in the carbohydrate tolerance.

Acromegaly

Acromegaly is a disease most common in middle life, characterized by certain nervous conditions and very marked enlargement of the hands, feet, and face, and frequently gigantism with kyphosis.

Etiology.—The cause of this disease is unquestionably disease of the pituitary body, the so-called hypophysis. The disease of this gland may amount to a veritable tumor, which can be discerned by x-ray, showing enlargement of the sella turcica.

It is due to hyperpituitarism, as shown by decreased carbohydrate tolerance, headache, etc., but when it is recognized by the symptoms above stated, the gland, in the great majority of cases, is in a state of inactivity. This is shown by tendency to adiposity and increased carbohydrate tolerance.

Symptoms and Physical Signs.—The symptoms of the disease are often brought to notice by the very marked enlargement of the hands, feet, and face. In the latter the spacing of the teeth is a marked feature. The enlargement of the hands consists in elongation of the fingers and increase in their circumference, also in enlargement of the joints (Fig. 99). In enlargement of the bones of the face, not only the bones are enlarged, but the soft parts as well.

The other symptoms which may occasionally accompany and sometimes precede these typical lesions are headache, vertigo, vomiting, mental irritability and other alterations, decrease of carbohydrate tolerance due to hyperpituitarism. There are later disturbances of the special senses—sight, hearing and taste, and of the cranial nerves—optic neuritis, oculomotor, and other paralyses. In addition there may be paresthesia, more or less severe pain, etc., in the extremities.

The skin may be harsh, dry and thickened, or there may be profuse sweating; sometimes the mucous membrane becomes thickened; the muscles may become atrophied; the huge frame of the individual contrasts markedly with the muscular weakness. Sexual disturbances are common in men and amenorrhea in women. These latter symptoms are the result either of a tumor of the gland or the loss of its function.

Particular attention should be given to arteriosclerosis and any hypertrophy and dilatation of the heart.

Conditions to be Differentiated from Acromegaly

The disease may be mistaken for:

Osteitis deformans
Osteo-arthropathy (both of the nerves and pulmonary type)
Diabetes mellitus.

OSTEITIS DEFORMANS.

Osteitis deformans may be quickly differentiated from acromegaly; in fact, the individual affected with osteitis deformans is usually past middle age, and it is the long bones which are affected first. These become thickened and bend; the individual becomes bow-legged and bow-armed, and is shorter than normal; the cranium becomes square and large and the face assumes a triangular appearance and often covered with bosses, which is in marked distinction to the large hands and feet, but relatively normal limbs, of acromegaly (See Fig. 104).

OSTEO-ARTHROPATHY.

In osteo-arthropathy the enlargement is at the joints, and frequently at the ankle joints and the wrist joints. The condition may be diagnosed by the presence of either grave nervous disease, such as tabes, or some grave pulmonary disease, such as tuberculosis, and sometimes bronchiectasis.

Diabetes mellitus.

In the early stages of acromegaly, while the gland is overactive, there may be polyuria and glycosuria. Cases of acromegaly may be mistaken for diabetes mellitus, but in the latter there are no skeletal changes, and, furthermore, x-ray of the skull will not show any changes in the sella turcica.

G. Infantilism

Etiology.—Infantilism, or dwarfism, and pseudo-obesity, are consequences of depressed pituitary function, but not every case of dwarfism is thus caused. Thus *cretinoid infantilism* is the result of defective thyroid function, and has been described on page 507. That variety of dwarfism known as *idiopathic infantilism* (so-called Lorain type) is of a different origin. The form of pseudo-obesity, known as *lipomatosis universalis asexualis,* is regarded as a form of hypopituitarism, and the same may be true of *Dercum's disease* (adiposis dolorosa).

Then there is a *cachectic infantilism* which is produced by any prolonged diseased state, such as hookworm disease, syphilis, chronic malaria and congenital heart disease. There is also said to be a *toxic infantilism* due to the prolonged effect of tobacco and alcohol; whenever this occurs sexual development ceases and physical development remains stationary.

So-called Lorain Type of Infantilism.—In this variety, as described by John Thomson, the figure resembles that of a child; when the patient is stripped, however, the outlines are found to be those of an adult, simply reduced. The head is proportionately small, the trunk well formed, the shoulders proportioned to the hips and the prominences of the bones and muscles are normally maintained. The subject is a miniature man (or woman), not one who has retained the characteristics of childhood beyond the proper time. There is no growth of facial, pubic or axillary hair, yet the genital organs, though small, are well shaped and proportioned to the size of the body. The intelligence in both sexes is generally normal.

The cause of this form is not generally known, but there is no reason to believe it is due to deranged pituitary secretion. It has also been called *"angioplastic infantilism"* because it is associated with defective development of the vascular system.

Pancreatico-intestinal Type of Infantilism.—Cases of infantilism associated with intestinal changes have been reported by Bramwell, Herter, Freedman and others. Bramwell considered the pancreas to be at fault, and his cases improved under treatment with pancreatic extract. In

Herter's case there were looseness of the bowels, often fatty stools, and a change in the flora of the intestine with a rise in ethereal sulphates in the urine.

Ateliosis (continuous youth) **and Progeria** (premature old age). Under these terms Gastings Gilford describes two types of dwarfs. Ateliosis also includes two subvarieties, the asexual and sexual. The *asexual variety* is an infantilism unassociated with cretinism, syphilis, or congenital heart disease, often more a delay than an arrest of development. In the *sexual form* there is a like delay in development until puberty, when the sexual organs mature and the body becomes set as a miniature man or woman. This is termed the "Tom Thumb" type of dwarf.

Conditions to be Differentiated from Infantilism

This condition can be confused with:

Cretinism
Diseases of the pituitary gland
Achondroplasia.

CRETINISM.

In cases of cretinism the individual is mentally deficient; there is lack of hair; the tongue is large, saliva dribbles from the mouth. The administration of thyroid gland preparation early in life causes miraculous and entire relief. Thyroid extract has no effect on infantilism of pituitary origin.

DISEASES OF THE PITUITARY GLAND.

The small individual, whose lack of development is due to hypopituitarism, is fat, infantile as to genitals and actions. The carbohydrate tolerance is greatly increased.

ACHONDROPLASIA.

Bowed legs, trident hands, short arms, pug nose, and marked mental activities are characteristic of achondroplasia; kyphosis and protuberant abdomen are also symptomatic.

Section XI

Diseases of the Locomotor System

A. Diseases of the Muscles

1. Myositis

(Inflammation of the Muscles)

This condition may be primary; when secondary it follows various infective processes, such as septic pneumonia, gonorrhea, septic endocarditis—indeed any serious infectious disease may be followed by suppurative foci, the result of the localization of their causative infecting agent.

(*a*) *Primary Suppurative Myositis*

Etiology.—Primary suppurative myositis is in reality an acute suppurative process of infective origin. The infecting focus may be an abrasion from one of the internal organs, from a small cutaneous pustule, indeed, from any area of suppuration.

Symptoms and Physical Signs.—Its symptoms are sudden; onset by chill and fever, pain in the muscle affected, sometimes ill defined, sometimes quite sharply localized. The local signs are simply the signs of a local inflammation from any cause—induration, heat, swelling, tenderness and often redness, as pus forms and fluctuation is experienced.

Conditions to be Differentiated from Primary Suppurative Myositis

It can be confused with:

Ostitis
Periostitis in which the muscle has become involved.

OSTITIS OR PERIOSTITIS.

Ostitis or periostitis, or any affection of the bone, can be discerned before operation by the use of the x-ray, when a shadow can be seen or rarefaction of the bone is evident. At the time of operation, careful exploration by means of the operator's finger will decide whether the bone or its covering is involved in the suppurative process.

This condition may easily, but without reason, be diagnosed as grippe or rheumatism. However, the constant fever, leukocytosis, induration of the affected part and fluctuation, will easily differentiate myositis from these two conditions.

(*b*) *Non-suppurative Myositis*

Non-suppurative myositis is a disease of *unknown origin,* though various theories as to the causative origin—parasitic, bacterial and toxic —have been advanced. At autopsy the muscles are pale, swollen and infiltrated with serum; at times hemorrhages are seen between the fibers.

Pain of a vague character at first soon becomes localized and severe; practically all of the muscles become implicated; the fever rises to 104° F. General edema occurs which is not symmetrical; the flexor and extensor surfaces are most involved; sometimes there is pitting due to subserous induration. There may be marked stomatitis and sore throat.

Conditions to be Differentiated from the Non-suppurative Myositis

The condition can be confused with:

Trichiniasis
Neuromyositis
Suppurative myositis
Syphilitic myositis
Scleroderma.

TRICHINIASIS.

Trichiniasis is characterized by gastro-intestinal symptoms, by painful muscles, by painful areas in any muscle, by fever, similar to the temperature of typhoid fever, and by a marked leukocytosis and eosinophilia.

NEUROMYOSITIS.

Neuromyositis can be diagnosed by the presence of a true neuritis in the affected parts.

SUPPURATIVE MYOSITIS.

Suppurative myositis can be differentiated by leukocytosis, great fever, localized areas of indurated, suppurating muscle tissue, and the presence of a local focus of origin.

SYPHILITIC MYOSITIS.

In syphilitic myositis there is a history of syphilitic infection; a Wassermann reaction is present.

SCLERODERMA.

Scleroderma is recognized by the localization of the induration in the skin; there is no fever; there is less disturbance of locomotion. This condition most commonly affects the skin of the neck and face.

(*c*) *Primary Myositis fibrosa*

This condition is characterized by chronic inflammation of the muscles, beginning in the lower extremities. The muscle fibers atrophy, and are replaced by fibrous tissue.

Symptoms.—The symptoms are pain—slight at first—affecting a single muscle or group of muscles; soon the patient is bedridden and contractions occur. There is no fever.

Conditions to be Differentiated from Primary Myositis fibrosa

These cases are mistaken for:

Myositis of other varieties
Arthritis deformans.

MYOSITIS OF OTHER VARIETIES.

In myositis, especially the suppurative cases, the skin of the legs is involved.

ARTHRITIS DEFORMANS.

The possibility of a diagnosis of primary myositis fibrosa as against arthritis is at once excluded by the fact that in arthritis the lesion is in the joints, and not in the muscles.

(*d*) *Progressive Myositis ossificans*

Cause.—The cause of this condition is unknown; cases are rare. The ossified men of museums are usually cases of this sort.

Characteristic Features.—Progressive myositis ossificans is characterized by the formation of bony masses in the muscles and fasciae, and indeed in all parts of the muscular system affected with inflammation.

The hardened areas are true bone, according to Munchmeyer, who studied the cases in 1864; the formation can be seen in all stages of growth, and many of these cases for years are diagnosed as rheumatism.

There is pain, swelling, induration and edema, the areas of induration occurring first in one muscle and then in another. The muscles of the back and neck are first affected. The evident myositis does not recover; the area becomes the seat of fibrous, followed by bony deposits. Exostoses sometimes occur.

Conditions to be Differentiated from Progressive Myositis ossificans

This condition can be mistaken for:

Arthritis deformans
Locomotor ataxia.

ARTHRITIS DEFORMANS.

In arthritis deformans, and especially the form of that disease which affects the intervertebral joints—*spondylitis*—the whole spine becomes fixed, the joints are involved—not the muscles as in myositis ossificans.

LOCOMOTOR ATAXIA.

Locomotor ataxia with arthropathy might be mistaken for progressive myositis ossificans, but the presence of signs of change in the spinal cord will decide the diagnosis.

2. Myalgia

Myalgia is a painful affection of the muscles, usually acute, at times chronic. It is often named according to the group of muscles affected—(a) lumbago, (b) torticollis, (c) pleurodynia, etc.

It is constantly mistaken for muscular rheumatism by the laity as well as by the physician, but in reality does not resemble rheumatism except by the presence of pain.

(*a*) *Lumbago*

Lumbago is a painful condition of the muscles of the lower erector spinæ group, often occurring suddenly—sometimes coming on after over-exertion. It may follow a severe wetting and exposure to cold while not properly protected.

Symptoms.—The symptoms are great pain and soreness of the muscles of the lumbar region. The patient cannot rise from a prone or sitting position without much difficulty and pain, but is perfectly comfortable while remaining quiet. There is no fever, no leukocytosis; considerable tenderness is felt over the affected muscles.

Conditions to be Differentiated from Lumbago

The condition can be confused with:

Renal colic
Pain in the lumbar region from acute nephritis
Perinephritic abscess
Uterine disease
Caries of the spine
Traumatism
Ileosacral subluxation
Lumbar neuralgia
Arthritis.

Renal Colic.

The pain in renal colic is unilateral and of extreme severity; it is conducted along the groin into the testicle and penis. There is blood in the urine, with leukocytes and often crystals.

Pain in Lumbar Region from Acute Nephritis.

Nephritis is occasionally, though not frequently, accompanied by pain in the back. The pain is under the lower ribs—further up than in lumbago, in which it is far down in the muscles. Albumin and tube casts can always be found in the urine. Almost all laymen believe pain in the back is due to "kidney disease"; however, it is well known that pain in the back is rarely due to that cause.

Perinephritic Abscess.

A perinephritic abscess might be mistaken for lumbago, but the fever, leukocytosis and exquisite tenderness over the inflamed region will differentiate it.

Uterine Disease.

Pain due to uterine displacements in the female can be diagnosed by examination of the pelvis. An abnormal condition of the uterus or adnexa will be found.

Caries of the Spine.

Caries of the spine can be diagnosed by tenderness over the spine, fixation of the spinal column, fever and leukocytosis. If a lumbar or psoas abscess occur, fluctuation will be present.

TRAUMATISM.

In traumatism there will always be the history of lifting a heavy weight or of some other sudden strain or fall affecting the back. Usually there is much tenderness over the muscles.

ILEOSACRAL SUBLUXATION.

Ileosacral subluxation or actual inflammation of the ileosacral joints can be diagnosed by x-ray examination of the joint, by tenderness of the joint and by discovering motion in the usual fixed articulation.

Frequently the sudden attacks of pain which come on after stooping or some such simple movement, or in the midst of perfect health—are due to a subluxation—and not to lumbago.

Finally this condition can be differentiated by the fact that fixation of the joint by adhesive strips or a proper bandage will give instant relief.

LUMBAR NEURALGIA.

Lumbar neuralgia, or a neuritis, can be differentiated by tenderness along the lumbar nerve; usually it is unilateral.

ARTHRITIS.

Arthritis affecting the lower thoracic or the lumbar spine can be differentiated by tenderness over the spinal column, fixation of the spine less marked than caries, and differentiated from the latter by the absence of emaciation, fever and leukocytosis.

(*b*) *Torticollis*

Torticollis is a painful condition of the trapezius and sternomastoid muscles. As stated above, this is a condition due to myalgia; the term is also applied to an inflammation of the spinal accessory nerve.

It occurs very suddenly in individuals who have been subjected to a sudden wetting followed by exposure, and particularly when the side and back of the neck have been subjected to sharp wind.

Characteristic Features.—It is characterized by an extremely painful condition of the muscles of the back and side of the neck. The head is drawn backward, the chin to the opposite side from that on which the muscles are affected. It is impossible to move the head independently of the body; hence when a person desires to look to one side or the other he has to turn his entire body around.

Conditions to be Differentiated from Torticollis

Disease of the spinal accessory nerve
Caries of the spine.

DISEASE OF SPINAL ACCESSORY NERVE.

Torticollis due to myalgia may simulate a disease of the spinal accessory nerve, causing the same position of the head, but which may be differentiated by the fact that the nervous condition is of long standing and is accompanied by actual atrophy of the muscles involved; also by the fact that in the spasmodic cases, the muscle may go into a tonic spasm and the head and chin be held exactly in the same position as in cases of myalgia. Here again, the disease is of long standing, and it is often interrupted by clonic convulsions of these muscles due to irritation of the nerve. The head is jerked violently from one side to the other, entirely without any control on the part of the patient himself.

Rarely do patients recover from these nervous conditions, whereas the cases of myalgia last only a few days.

CARIES OF THE SPINE.

Caries of the spine might possibly be mistaken for torticollis of muscular origin. In caries of the spine there is extreme ill health; the neck remains in a fixed position, and the least motion of the head causes extreme pain. There is tenderness over the spine, also fever, and there may be tubercular deposits in other parts of the body.

(*c*) *Pleurodynia*

Pleurodynia is a painful affection of the intercostal and serratus muscles, and at times of the pectoral muscles.

It is characterized by an extremely painful state of the muscles of the side affected. There is pain on pressure, on breathing, and upon motion.

Conditions to be Differentiated from Pleurodynia

The conditions with which it is confused are:

Intercostal neuralgia
Pleurisy
Caries of the spine
Spondylitis.

INTERCOSTAL NEURALGIA.

Intercostal neuralgia is often difficult to distinguish from pleurodynia, the pain being of about the same character, but with this difference: in intercostal neuralgia the pain is along the line of the nerve affected and is particularly marked at the point of exit or at the nerve posteriorly, in the mid-line and anteriorly, while in pleurodynia the tenderness extends over the whole side.

PLEURISY.

Pleurisy can at once be differentiated by the presence of fever and leukocytosis; a friction sound can be developed when the patient takes a long breath, which is not the case in pleurodynia.

CARIES OF THE SPINE—SPONDYLITIS.

Here again disease of the spine, caries, and possibly spondylitis due to infection, might be mistaken for pleurodynia, but both of these conditions so evidently are connected with disease of the spinal column itself that the diagnosis is readily made. The x-ray will be of the greatest value. There is no change in the intervertebral joints in pleurodynia.

3. Myotonia

(*Thomsen's Disease*)

Myotonia is a tonic spasm of the muscles, felt when movement is attempted. Cases are usually hereditary.

Occurrence.—According to Osler, all cases have occurred in family groups. It is more common in males than in females, appearing first when the person is about twenty years of age. According to Steiner, if it manifests itself earlier, no attention is paid to it by friends and other observers because of the vagueness of the symptoms.

Physical Signs.—Attention is attracted by the fact that the individual is stiff in his movements. Although the child is able to play he is prevented from continuing on account of recurring contraction of the muscles. In walking he becomes stiff, but after a few steps he is able to walk without much difficulty, but if the muscular act be interrupted there is a recurrence of the symptoms when the motion is again undertaken. The muscles appear to be hypertrophied but lack power; there is no pain.

Steiner describes the condition as follows:

> "The electrical reactions have been found characteristic of the myotonic disorder, and have been collectively designated by Erb 'the myotonic reaction.' They are as follows: (1) The motor nerves show no increase of irritability to mechanical stimuli; (2) to the faradic current the motor nerves are quantitatively normal, but if the current be strong, the contraction produced on closing the current lasts much longer than it does in health; (3) to the galvanic current the motor nerves are quantitatively normal; but here also if the current be strong, the contraction lasts longer than in health; (4) mechanical stimuli applied to the muscles, as by hitting them, produce contractions more easily than in health; (5) the faradic current applied directly to the muscles, if strong, sets up a contraction which lasts from five to thirty seconds; (6) when the galvanic current is applied directly to the muscle, K.C.C. and A.C.C. are equally easy to obtain; while in health, as is well known, K.C.C. is more easily elicited than A.C.C. In Thomsen's disease,

even with weak currents, the contraction lasts longer than in health; with strong currents it sometimes lasts some seconds and relaxes very slowly. With the stabile application, well-formed, wavelike contractions are seen to proceed slowly from the cathode to the anode (Hale White). All of the reported cases have not shown an exact resemblance to Erb's cases, which gave the myotonic reaction as Hale White thus described it. They have in almost every instance though revealed a normal mechanical faradic and galvanic excitability of the motor nerves, but an increased mechanical, faradic, and galvanic excitability of the muscles. The peculiar rhythmical, vermicular contractions have only been obtained in occasional cases by the application of a strong and steady galvanic current. Erb subsequently did not attach much importance to them, but thought they could be obtained in every case by proper manipulations.

"The tendon reflexes vary, being increased, normal, or absent. In two of Jacoby's patients the knee jerks seemed at first exaggerated, but became weaker and weaker, after successful elicitations, until no response was obtainable. The knee jerks later reappeared after a short interval of rest. There are generally no sensory disturbances, with the exception of rare paresthesia. The quantitative urine examinations have yielded inconstant results."

Morbid Anatomy.—The morbid anatomy is not well known. The disease is incurable. Neither the acts of swallowing, respiration or micturition are affected. In light cases the patient can conceal the condition; in severe cases, however, the patient appears to be "chained to the floor" on attempting to walk.

Conditions to be Differentiated from Myotonia

Thomsen's disease may be mistaken for the following conditions:

There are certain individuals who have INCREASED REACTIONS TO MECHANICAL AND ELECTRICAL IRRITANTS, but these are acquired; they are not hereditary, and they do not have the myotonic reaction.

In PARAMYOTONIC CONGENITA there is not the myotonic reaction, and the condition only appears on the application of cold.

There are also cases reported of MYOTONIA CONNECTED WITH TETANY, AND WITH MULTIPLE NEURITIS, but these cases are not congenital, and there is no myotonic reaction.

4. Paramyoclonus multiplex

This condition is described originally by Friedreich. Osler in his article defines it as "a clonic contraction, chiefly of the muscles of the extremities, occurring either constantly or in paroxysms."

Any of the muscles of the body except the eye muscles may be affected. The muscles affected are thrown into rapid, violent contractions, as though irritated by an electric current; there is no fibrillary tremor. In the violent attacks the body is tossed about. There is no disturbance of sensation or to the electrical response; there is no atrophy of the muscles.

Conditions to be Differentiated from Paramyoclonus multiplex

According to McCarthy, the condition may be mistaken for:

Myoclonus of functional or hysterical type
Myoclonus of the convulsive type
Myoclonus of the degenerative chorea or familial or epilepsy type
Myoclonus of the infectious and symptomatic choreas.

MYOCLONUS OF EPILEPSY TYPE.

The hysterical muscular spasms may be differentiated by their irregularity, by the presence of other hysterical spasms, by their inclination to increase on excitement, and by the other conditions which increase the evidence of hysteria generally.

MYOCLONIA OF THE CONVULSIVE TYPE.

In *convulsive tic* the spasms are purposeful; there is quick spasm of the orbicularis, of the muscles of the face, of the muscles of the shoulder.

MYOCLONUS OF EPILEPSY TYPE.

Myoclonus epilepsy is distinguished by the fact that in this condition there are attacks of distinct epileptic seizures.

INFECTIVE CHOREAS.

The infective choreas are characterized by the purposeless movements involving large muscle groups; the movements are not so quick, and do not resemble the response to an electric shock.

5. Myasthenia gravis

McCarthy's article in "Modern Medicine" embraces the findings in 182 cases. He defines the condition as one with fatigue symptoms referable to the muscular system, due to an exhausted condition of nervous innervation without any changes in the nervous system, but with minor changes in the muscles and with lymphocytic infiltration in the muscles.

Symptoms.—The symptoms are decided fatigue in the muscles after a single action—if the muscular activity is continued an actual paralysis occurs. After a period of rest the muscles regain their tone; after a night's rest the muscles seem about in a normal condition, though in the elevation of the eyelids and the muscles of the jaw there is some weakness.

The disease begins slowly, the eyelids showing a tendency to droop as the day advances, and finally complete ptosis may occur. This is usually bilateral, but it may be greater on one side than the other. After a period of long rest the eyelids may become almost normal.

Diplopia occurs as the result of weakness of the ocular muscles. Sometimes there are jerky movements of the eyes due to irregular action of these muscles which closely resembles true nystagmus. Reading becomes difficult on account of loss of muscle balance.

When the patient has bilateral ptosis, the drooping of the lids is compensated by a throwing back of the head; if the muscle of the head becomes affected, this compensation does not occur.

Involvement of the muscles of deglutition and of the laryngeal muscles often causes inability to chew, to swallow and to speak. Finally all the muscles of the body may become affected, and the patient be invalided. Power is at first regained after rest.

The so-called myasthenic reaction consists in the fact that if a strong interrupted current be applied there is at first good reaction which soon decreases and disappears. After an interval of two minutes, a response is again present; this again disappears—and more rapidly than at first.

Conditions to be Differentiated from Myasthenia gravis

The following may all be mistaken for myasthenia gravis:

Bulbar palsy
Pseudobulbar palsy
Diphtheritic paralysis
Polioencephalitis superior
Muscular dystrophies
Hysteria
General asthenia.

BULBAR PALSY.

Bulbar palsy affects first and preferably the muscles of the tongue and deglutition; the eye muscles are rarely affected, and only in advanced stages. There is atrophy and fibrillating tremors.

PSEUDOBULBAR PALSY.

Pseudobulbar palsy is characterized by repeated apoplectic attacks.

DIPHTHERITIC PARALYSIS.

In diphtheritic paralysis there is the history of antecedent diphtheria; there is rapid onset, the palatal muscles often being first affected. There is no electrical response as there is in myasthenia gravis.

Polioencephalitis Superior.

Polioencephalitis is an acute inflammatory disease. The ocular muscles are paralyzed and bear no relation to fatigue.

Muscular Dystrophies.

Muscular dystrophies do not have the myasthenic reaction.

Hysteria.

Hysteria can be differentiated by the general hysterical state, by increase to suggestion, and by excitement, and by the absence of electrical response of myasthenia gravis.

General Asthenia.

The history of the disease causing the asthenia is present in asthenia due to prolonged systemic disease.

None of the above have the myasthenic reaction.

6. Amyotonia congenita

(*Oppenheim's Disease*)

Characteristic Features.—A congenital affection consisting in atrophy with corresponding weakness of muscles (hypotonia and atonia), especially of the extremities, most marked in the lower extremity; it is associated with loss, more or less complete, of the tendon reflexes. In advanced cases the weakness resembles paralysis, but closer observation discovers feeble contractions in the muscles, but not sufficient to move the limbs. The muscles of the trunk and neck are most rarely affected, while those of the eye, tongue and throat escape, as does the diaphragm, while the intercostal muscles may be invaded. The electrical reaction is affected proportionally to the hypotonia. Intelligence and sensation are undisturbed. Neither hereditary nor family tendency seems to play any part. Although always congenital the symptoms are not always noticed immediately after birth. Oppenheim, who was the first to investigate the disease, believes the morbid change is in the muscles which are arrested in their development, and thinks it has no relation to muscular dystrophy. He admits the possibility of disease in the cells of the anterior horns of the cord, whence it is however, the latter developing acutely in a previously normal child.

Diagnosis.—"Congenital amytonia differs from progressive muscular dystrophy especially in the absence of family tendency, in being congenital, and in the absence of progressive increase in the symptoms; from amaurotic family idiocy in that in the latter the symptoms increase and the ophthalmological changes are pathognomonic. It occurs in more than one member of a family."

7. Family Periodic Paralysis

Etiology.—Family periodic paralysis is a rare disease of unknown pathology, which attacks members of different generations of a family. Investigations seem to show that it is a disease of metabolism which acts in some way upon the muscles themselves.[1]

Symptoms.—In the majority of cases the symptoms first appear between the ages of ten and twenty. The attacks in some cases tend to appear after severe muscular exertion. They may or may not be preceded by prodromata, as feelings of weariness, numbness, headache, backache, sweating, etc.

Loss of power often appears first at night, and the legs are usually first affected, followed by involvement of the muscles of the arms, trunk and neck. The muscles supplied by the cranial nerves are rarely affected and in some cases the weakness may be confined to the legs.

The symptoms reach their height in from a few hours to several days, the duration of the attack being from ten to forty-eight hours, after which recovery gradually occurs.

During the attack the deep reflexes are lost, the electrical reactions vary from quantitative decrease to absence of response to either the constant or induced current, and in some cases there is cardiac enlargement with a mitral systolic murmur.

In the interval between the attacks the reflexes, electrical reactions and cardiac sounds are normal.[2] There are no sensory symptoms and the mental state is normal.

Differentiation.—This disease must be distinguished from MYASTHENIA GRAVIS.

In no other disease than periodic paralysis is there a similar history, i. e., attacks of paralysis without apparent cause, disappearing in a short time to return later. In myasthenia gravis motor weakness develops after muscular exertion, which disappears after a short rest. The weakness is confined to the muscles or limb used, and occurs invariably after exertion during the course of the disease. It is not a family disease and the electrical reactions are peculiar (See Myasthenic Reaction, page 523).

In periodic paralysis during the interval, exertion has no influence.

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[1] Observations on a Case of Family Periodic Paralysis, Edsall & Means, Am. J. Med. Sci., Aug., 1915, p. 169.

[2] Unless there is valvular disease due to endocarditis from some other cause.

8. Progressive Muscular Dystrophies or Myopathies

Progressive muscular dystrophies, or myopathies, are the terms applied to a hereditary familial condition characterized by progressive muscular wasting, in some cases preceded by apparent hypertrophy, which first appears in early life and is due to degeneration of the muscle fibers,

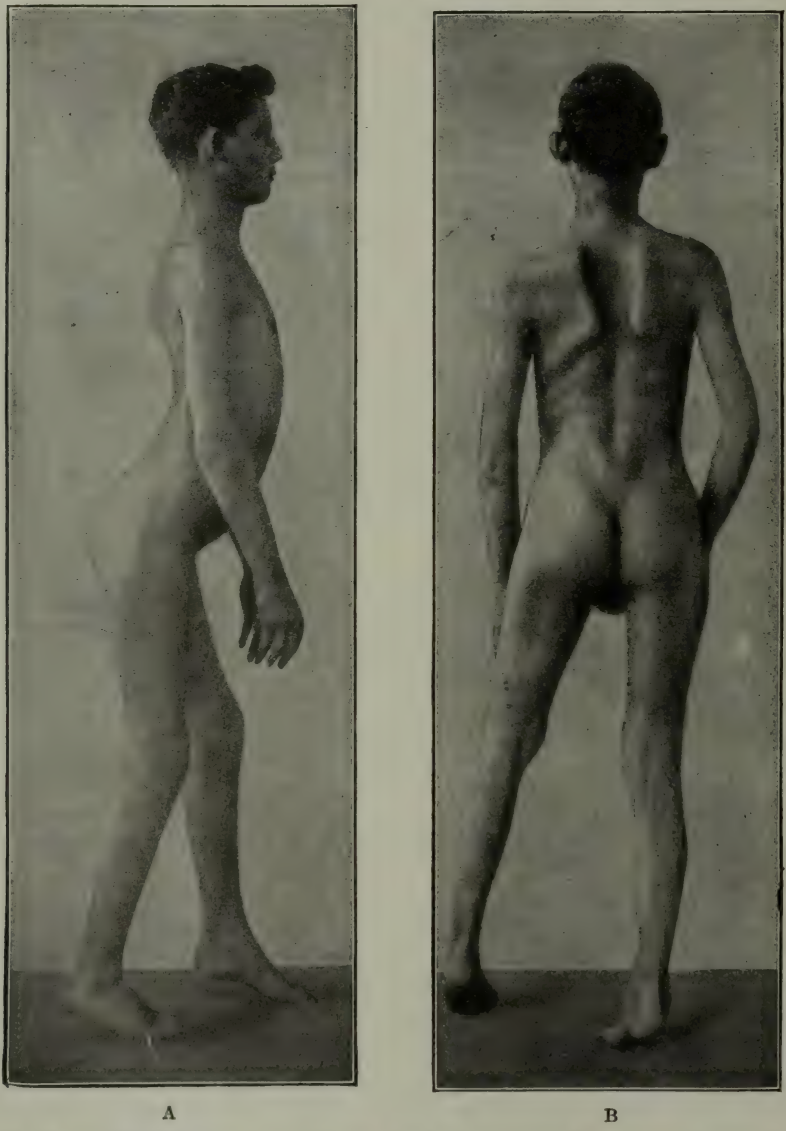

Fig. 102 (A and B).—Lordosis and Typical Gait in Juvenile Dystrophy. (After Heinrich Curschmann, "Textbook of Nervous Diseases," published by P. Blakiston's Sons & Co., Philadelphia.)

without involvement of the nervous system. They are abiotrophies (page 686). A number of clinical types have been described dependent upon the age at onset, muscles first affected, and the occurrence or not of pseudo-hypertrophy.

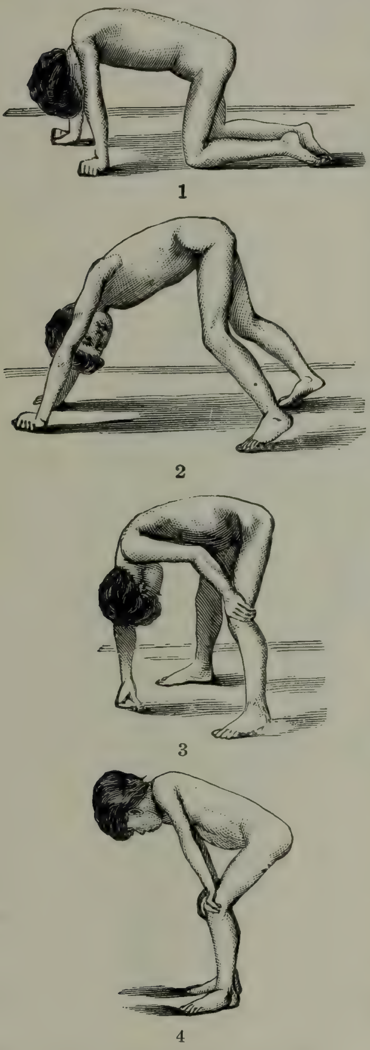

Fig. 103.—Positions of a Child with Hereditary (Pseudohypertrophic) Muscular Atrophy, on Arising to an Erect Attitude. (After Gowers.)

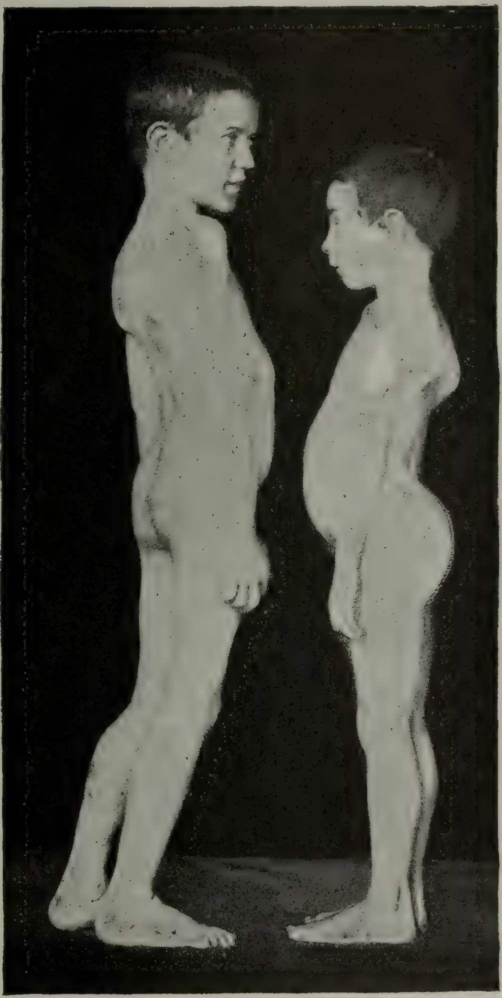

Fig. 104.—Pseudomuscular Hypertrophy in Brothers. (Infirmary of Nervous Diseases, Philadelphia.)

Types.—These types may, however, more or less overlap, and some cases may later develop degeneration of the anterior horn cells and symptoms characteristic of spinal muscular atrophy (page 694).

The more important types are:

(*a*) Pseudomuscular hypertrophy.
(*b*) Erb's juvenile type or scapulohumeral type
(*c*) Facioscapulohumeral type (Landouzy and Déjerine).
To these may possibly be added:
(*d*) Myotonia atrophica
(*e*) A true hypertrophic type.

Symptoms.—Certain symptoms are peculiar to all types: they appear usually in childhood; several members of a family and in different generations may be affected; the weakness and atrophy affects the proximal muscles first; fibrillary tremors and reactions of degeneration are absent; the deep reflexes become diminished and finally lost; the progress is usually slow; no sensory symptoms occur; and the limbs are usually cold and cyanotic. Enlargement of the parotid, salivary, and other glands has been observed in some cases.

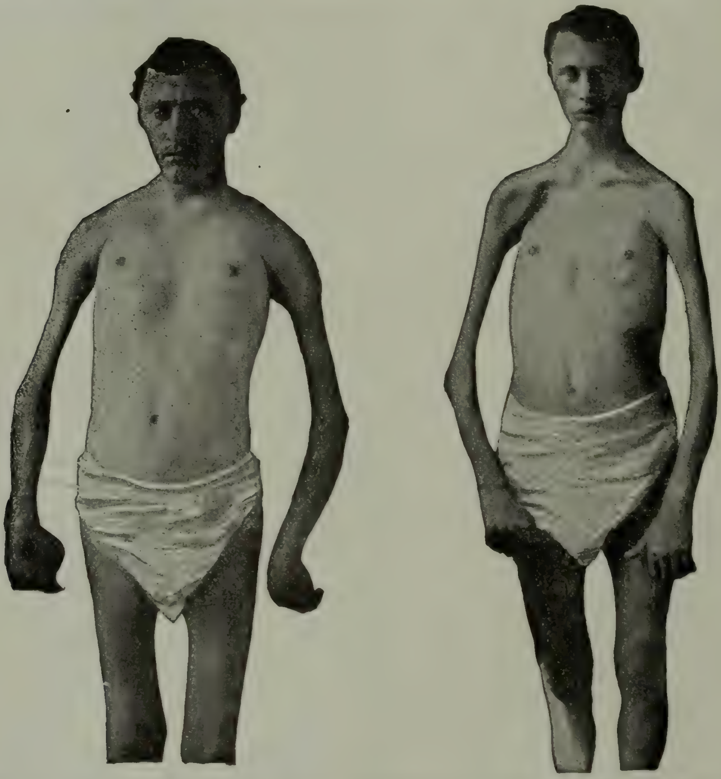

Fig. 105.—Two Brothers with Juvenile Muscular Atrophy. (After Strümpell.)

(*a*) PSEUDOMUSCULAR HYPERTROPHY.

Occurrence.—Pseudomuscular hypertrophy usually appears under the age of ten, often shortly after the child begins to walk.

Symptoms.—Weakness is first noticed in the legs, shown by a tendency to fall and a peculiar waddling gait, much like that of a duck, shown in Figures 102 A and B. It will also be noticed that the child has difficulty in arising from the recumbent to the erect position. This is done in a characteristic manner, shown in Figure 103, the patient actually climbing up his legs. If the legs are examined the calf muscles will appear to be unusually large and firm. The firmness, however, has not the elastic feel of true muscle, but resembles that of a ball of wood (Fig. 104). Other muscles, viz., the glutei, deltoids, lumbar muscles, triceps, and the infraspinati, may also become apparently hypertrophied. The strength, however, will be found to be much diminished. Later the latissimus dorsi, pectorals, upper arm—except those mentioned above—and thigh muscles atrophy. Eventually the hypertrophied muscles may also waste. When the patient stands there is marked lordosis, as shown in Figure 102, due to weakness of the back muscles.

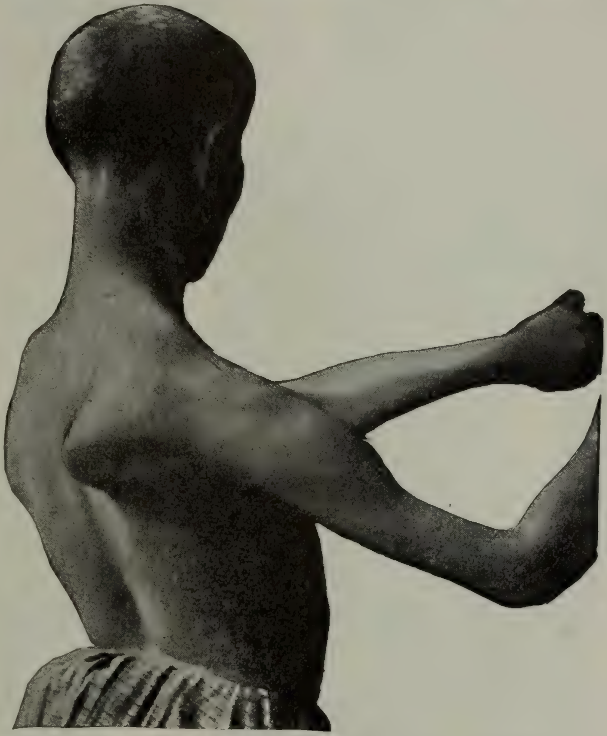

Fig. 106.—Juvenile Muscular Dystrophy. Prominence of the Scapulae When the Arms Are Raised in Consequence of Atrophy of the Serratus and Rhomboidei. (After Strümpell.)

(*b*) Scapulohumeral or Erb's Type.

Occurrence.—This type may develop somewhat later in life.

Symptoms.—In the scapulohumeral or Erb's type the shoulder muscles are first attacked, then those of the upper arm, back, pectorals, latissimus dorsi, trapezii and then those of the thigh. The muscles of the forearm and leg may not suffer at all, or else do so later. The marked atrophy of the proximal muscles, while the distal ones are fairly well developed, is characteristic (Figs. 105-107).

The gait and method of arising from the recumbent position are similar to those found in pseudo-muscular hypertrophy.

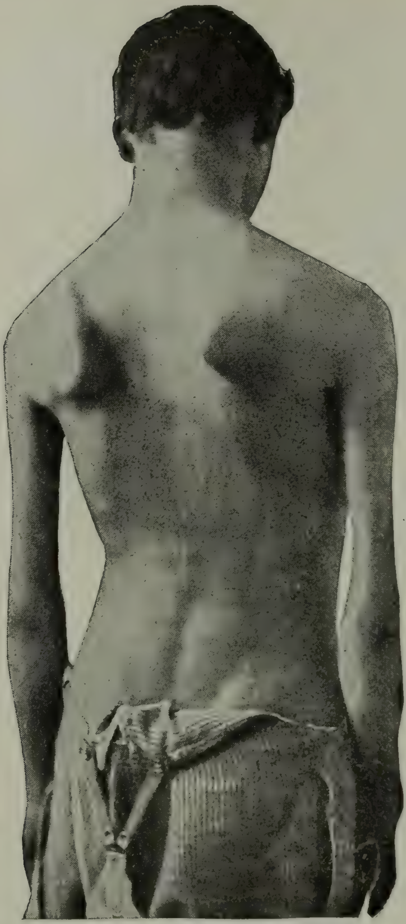

Fig. 107.—Muscular Dystrophy. Prominence of the Scapulae When the Arms Are Hanging Down. (Erlangen Medical Clinic.)

(*c*) Facioscapulohumeral Type.

Occurrence.—The facioscapulohumeral type usually develops quite early, often at three or four years of age.

Symptoms.—The atrophy begins in the muscles about the angle of the mouth, causing a protrusion of the lips which has been termed the myopathic face or "tapir mouth" (Fig. 108). The subsequent course is similar to that of the scapulohumeral form.

(*d*) Myotonia atrophica.

Symptoms.—In myotonia atrophica the appearance of the face is similar to that of the facioscapulohumeral type.

In addition there is atrophy of the sternomastoids, vasti muscles of the thigh, and dorsal flexors of the feet. Sometimes the masseters, temporals and muscles of the forearm are affected. In addition to the atrophy there is myotonia of certain muscles, shown by their slow relaxation after they are contracted

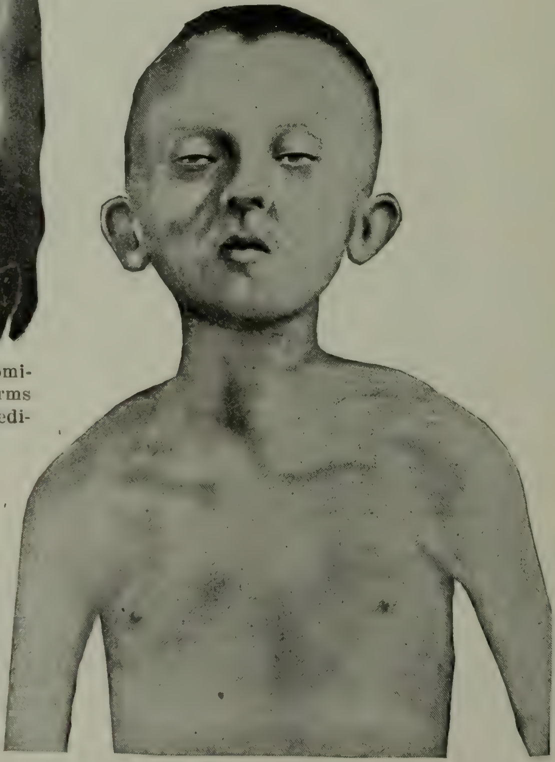

Fig. 108.—Juvenile Myopathic Muscular Atrophy in a Ten-Year-Old Child, with Marked Implication of the Facial Muscles. Inability to Close the Eyes or Move the Lips. Atrophy of the Pectorals, etc. (From the Erlangen Medical Clinic.)

(See Thomsen's Disease). Cataract is frequently associated in these cases.

(*e*) TRUE HYPERTROPHY OF THE MUSCLE FIBERS.

Cases have been described in which there was muscular weakness associated with true hypertrophy of the muscle fibers, as distinguished from the pseudohypertrophy due to increase of connective and fatty tissue which occurs in the type of that name. In these cases there was no atrophy of any muscles, the symptoms were noticed later in life, and muscular examination of a piece of muscle showed the nature of the hypertrophy.

Conditions to be Differentiated from Progressive Muscular Dystrophies

Dystrophies must be distinguished from:

Amyotonia congenita
Progressive neurotic muscular atrophy
Progressive spinal muscular atrophy
Acute anterior poliomyelitis
Multiple neuritis
Obstetric paralysis.

AMYOTONIA CONGENITA.

Amyotonia congenita, which is sometimes classed among the dystrophies, is either congenital or the symptoms develop in early infancy. It is characterized by marked hypotonicity of the muscles, but there is no wasting. It is not a family disease and improvement often occurs.

PROGRESSIVE NEUROTIC MUSCULAR ATROPHY.

In progressive neurotic muscular atrophy the atrophy begins in the small muscles of the feet, then of the hands. In other words, distal muscles suffer first and not the proximal. Fibrillary tremors and reactions of degeneration are present and sensory symptoms, as pain and slight diminution of sensation, may be present.

PROGRESSIVE SPINAL MUSCULAR ATROPHY.

Progressive spinal muscular atrophy usually appears in early adult life. The distal muscles are first affected and fibrillary tremors are prominent.

Werdnig-Hoffmann Type.—The Werdnig-Hoffmann type, which appears in infancy, and may be a familial disease, may be mistaken. In it there is atrophy beginning in the pelvic girdle, then the muscles of the hands, and progression, as in the adult type, with fibrillary tremors. Usually death occurs early.

	Dystrophies	Amyotonia Congenita	Progressive Neurotic Atrophy	Progressive Spinal Atrophy	Multiple Neuritis	Anterior Poliomyelitis	Obstetric Paralysis
Age at onset	Usually 2–10 years	Congenital or very early infancy	About 15 years	20–30 years except Werdnig-Hoffmann type which is in infancy	Any age	Usually 4–5 years. May at any age	At birth. Often not noticed until child begins to move arm
Heredity	Family disease	Is not a family disease	Family disease	May be rarely	Is not	Is not	Is not
Muscles first affected with atrophy	Proximal muscles and those of back. Pseudohypertrophy appears in calf muscle first	All muscles hypotonic but no atrophy or pseudohypertrophy	Distal muscles	Distal muscles. Pelvic girdle in Werdnig-Hoffmann type	Depends on nerves affected	Any muscles	Shoulder, upper arm, one side
Fibrillary tremors	None	None	Present	Present	None usually	None usually	None
Electrical reactions	No change until late, then quantitative decrease	May be. Quantitative decrease, especially faradic	Partial reactions early	Partial reactions early	D R or quantitative decrease	Same as neuritis	Same as neuritis
Sensory Symptoms	None	None	Slight pain. Diminution of sensation sometimes	None	Usually pain except when due to diphtheria or lead	None	May be loss of sensation in areas, supplied by affected nerves
Prognosis	Slowly progressive. Never recover	Often do recover	Slowly progressive	Progressive	Usually recover	Improvement may occur, not progressive, acute onset	Not progressive. Improvement may occur

ACUTE ANTERIOR POLIOMYELITIS.

In acute anterior poliomyelitis there will be a history of acute onset and constitutional symptoms. The resulting paralysis remains stationary and reactions of degeneration are present.

MULTIPLE NEURITIS.

Multiple neuritis is characterized by pain and tenderness over the nerve trunks, excepting possibly in the diphtheritic form, when a history of the previous existence of that disease and the improvement which usually occurs eliminates dystrophy.

OBSTETRIC PARALYSIS.

The upper arm type of brachial paralysis (obstetric paralysis), which is due to the injury to certain roots of the plexus at birth, is not progressive, and changes in the electrical reactions will usually be found.

B. Diseases of the Joints

1. Arthritis deformans

(*Osteo-arthritis, Rheumatoid Arthritis*)

Origin.—This is a progressive arthritis, occurring as the result of some infection, the source of which is often obscure.

Course of Disease.—It usually attacks the small joints first—the fingers and the toes; it affects the articular and periarticular tissues. It is progressive in character, finally affecting all the joints. The articular surfaces are destroyed and contractures appear. In the course of the disease the shape of the joints may be changed; bony outgrowths, Heberden's nodes, occur commonly at the phalangeal joints. Frequently the progressive character of the disease disappears leaving disabled joints, but the patient remains in good health.

Acute Form.—There is an acute form which has at first all the earmarks of an acute articular rheumatism, with fever, redness, and swelling of the joints. In these cases, however, the joints, instead of being restored as the acute symptoms disappear, become chronically affected, and one or more of the joints becomes useless; fixation and contractures of the joints occur.

It is always a polyarthritis; there are sometimes enlarged glands and enlarged spleen with fever. In certain cases the bones become involved, resembling the arthropathies, in others there is a bony ring about the joint.

In the cases in which the spinal column is affected, there is fixation of the column and great atrophy of the muscles of the shoulders and chest. The x-ray furnishes valuable early evidence of first changes in and about the bones, and finally of the bony outgrowths.

This disease is at once the despair of the pathologists and the clinician. It is constantly called "chronic rheumatism," with which it has little in common except pain and arthritis.

Conditions to be Differentiated from Arthritis deformans

It may be mistaken for:

Rheumatism
Gout
Septic arthritis
Gonorrheal, syphilitic and tubercular arthritis
Neuritis
Arthropathies.

ACUTE ARTICULAR RHEUMATISM.

Acute articular rheumatism usually begins in the large joints; one or a number of joints may become affected. There is great swelling, tenderness and redness, high fever, sweats and leukocytosis—the patient is extremely ill in these cases.

The joint rather suddenly becomes much improved, whereupon there is a change of the same nature in the general condition of the patient, to again become worse with the involvement of a new joint, until almost all of the joints of the body become involved. When the joint recovers, there is *not left behind any permanent loss of function of the joint.* This fact, to the author's mind, is the one distinctive characteristic feature of the joint of rheumatism. Rheumatism is favorably affected by the use of salicylates; arthritis deformans is not so affected.

Acute rheumatism frequently is accompanied or preceded by an acute tonsillitis; often the endocardium and pericardium are affected, whereas this affection is extremely rare in arthritis deformans. There is a close relation between rheumatism and chorea—none between chorea and arthritis deformans.

GOUT.

Gout, in its typical acute attack, begins by severe digestive disturbance, high fever, severe painful swelling of the joint of the great toe. It is common in the users of beer and ale, and in "high livers." In the chronic forms there are tophi about the various joints and along the cartilages of the ear. There is not the same progressive disturbance of the joints, which is so characteristic of arthritis deformans.

SEPTIC ARTHRITIS.

Septic arthritis is usually, but not always, monarticular. There is the history of acute septic condition, of which the joint affection is but a part; there is fever and leukocytosis.

GONORRHEAL, SYPHILITIC AND TUBERCULAR ARTHRITIS.

These conditions are usually monarticular. An x-ray examination of the joint will show early cartilage and bone changes; there is a history of the original disease. In chronic cases where the symptoms of the original disease have disappeared, the origin of the infection may be judged by the gonorrheal and syphilitic fixation tests and Wassermann reaction.

NEURITIS.

Neuritis, especially that affecting the shoulder joint, can be recognized by the evident inflammation of the nerve, by its being monarticular, and by the early *muscular* atrophy. X-ray will show no change in the bony articulation.

ARTHROPATHY.

Arthropathy, occurring as the result of locomotor ataxia or of chronic lung disease, can be diagnosed by the characteristic signs of locomotor ataxia and of chronic pulmonary state.

2. Intermittent Hydrarthrosis

Intermittent hydrarthrosis is a periodic swelling of one or more joints, without pain or stiffness and without fever. The attacks frequently are sudden in onset; a series of attacks may recur regularly. There may be an anginoneurotic edema.

Differentiation.—This condition must not be mistaken for AN ACUTE PHASE OF ARTHRITIS. All the other arthritides have other symptoms than the simple hydrarthroses; they are not intermittent as they are in hydrarthrosis—there is likely to be fever.

C. Diseases of the Bones

1. Hypertrophic Pulmonary Arthropathy

Hypertrophic pulmonary arthropathy is a condition characterized by enlargement of the ends of the long bones of the hands and feet and clubbing of the fingers occurring in diseases of the lungs.

There are usually no symptoms referable to the changes themselves.

The ends of the tibia and fibula especially become very large. The feet and hands enlarge, as do the nails, the latter becoming incurved.

Differentiation.—This condition may be mistaken for ACROMEGALY, however acromegaly has not the same enlargement of the terminal parts of the bones; the nails are not incurved, and there is no lung lesion.

2. Osteitis deformans

(Paget's Disease)

Physical Signs.—Osteitis deformans is an affection of the bones, chronic in character, with thickening and bowing of the shafts of the bones; it especially affects those of the femur, the tibia, the clavicles and the bones of the arm. The head gradually enlarges—due to thickening of the bones of the skull.

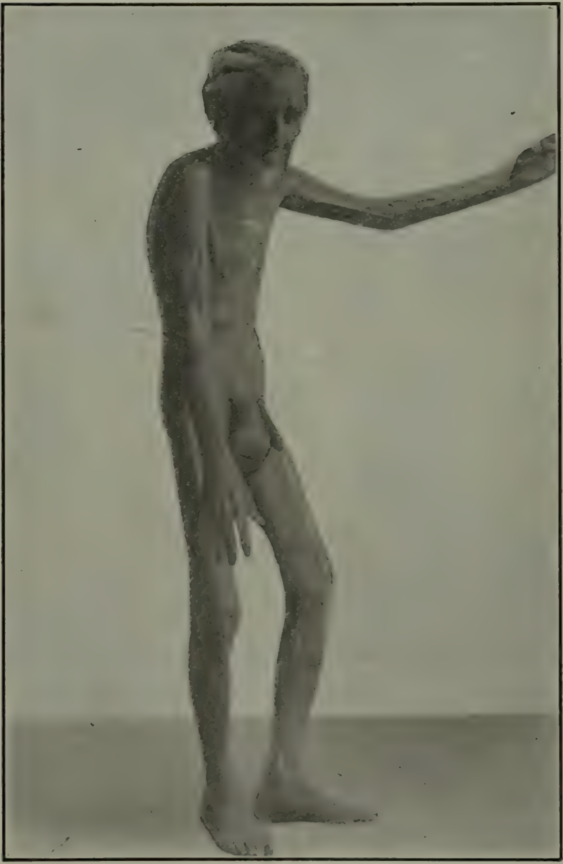

Fig. 109.—Osteitis deformans. (Original Observation.)

Course of Disease.—The disease affects elderly individuals — about sixty years of age — usually without symptoms which attract the patient's attention. The head enlarges, the individual becomes bow-legged, the spine becomes curved, and the individual gradually becomes shorter than when in health. Sometimes the patient complains of pains as in rheumatism.

Differentiation. — The condition might be confused with ACROMEGALY, but in the latter affection the bones are larger, and no rarefaction is shown by the x-ray. In acromegaly the teeth are spaced at greater distances than they are in osteitis deformans; there is evidence of sudden overgrowth of the bones.

3. Leontiasis ossea

This is a disease of adult life.

It consists in a thickening of the bones of the head, particularly of the cranium.

Differentiation.—It might be mistaken for OSTEITIS DEFORMANS, but there are no changes in other bones of the body; also it might be confounded with ACROMEGALY, but the latter is differentiated by the presence of enlargement in other portions of the bony framework.

4. Achondroplasia

Achondroplasia, a chondral dystrophy, has been known under the synonyms of "chondrodystrophia fetalis," "fetal rickets," "fetal cretinism" and "micromelia." Achondroplasia is of great antiquity. Parnet draws attention to the fact that at the British Museum there are a number of glazed earthenware images which are unquestionably models of achondroplastic individuals. They were represented as dwarfs with big heads, crooked legs, very long arms, etc.

Characteristic Symptoms.—The characteristic symptoms of achondroplastic individuals are the following:

(1) Short stature of adult achondroplastics (Fig. 110).

(2) Normal length of the trunk as compared with the short limbs.

(3) Marked bowing of both upper and lower extremities.

(4) Unusual prominence of the points of attachment of the muscles on the bones of both upper and lower extremities.

(5) Relatively similar length of all fingers. There is a peculiar separation of the second and third fingers at the second phalangeal joints, causing the fingers to spread—the so-called trident hand (Fig. 111).

(6) Depression of the base of the nose—"pug nose."

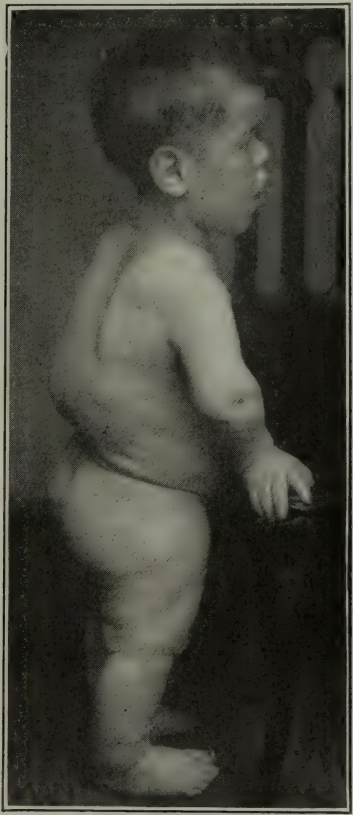

Fig. 110.—Photograph of Achondroplastic Boy, Aged 12 Years. (Personal Observation.)

(7) The vault of the cranium is unusually large, as compared with the base of the skull and the face.

(8) The pelvis is small.

(9) Lumbar lordosis is present without exception, as is the protuberant abdomen.

(10) The hair is soft and abundant in the normal situations.

(11) The intellect is normal.

(12) The genitalia and sexual instincts are normal.

(13) There is a tendency to superabundance of fat.

(14) The deformity is congenital.

(15) There is marked decentralization of the body. The umbilicus is always below the middle, while in normal individuals over one year, it is above the middle point.

Conditions to be Differentiated from Achondroplasia

The conditions with which achondroplasia can be confounded are:

Rickets
Cretinism
Congenital syphilis and osteogenesis imperfecta.

RICKETS.

Achondroplasia is a *congenital* disease; the lesions are complete at birth. The deformities present are but exaggerated with the growth of the individual. Rickets is a *postnatal* disease. The lesions differ entirely in the two affections and may at once be differentiated by the x-ray.

In achondroplasia the lesion is in the cartilage; the epiphyses are about normal; the enlargement at the ends of the bone is due to cuplike projections of the diaphyses. In rickets the enlargement at the ends of the bones is in the epiphyses itself; the enlargements forming bosses at the muscular attachments, present in achondroplasia, are not found in rickets. In achondroplasia the bones are hard, in rickets they are soft. The chest and trunk are normal in achondroplasia; they are affected in rickets. There is "pug nose" in achondroplasia which is absent in rickets; the vault is normal in achondroplasia and bossed in rickets; the bones affected in achondroplasia are those laid down in cartilage, while any of the bones may be affected in rickets. Achondroplasia is a permanent lesion; a patient with rickets may recover. Apert says, "An individual is born achondroplastic, but an individual may become rachitic and recover."

CRETINISM.

The points of differentiation from cretinism are the following:

A cretin lacks intelligence; this is in contrast to achondroplastics who have a normal or unusually bright intellect. The hair of the cretin is scarce and coarse, that of achondroplastics abundant and normal; the tongue of a cretin is protruded, and there is drooling; neither of these symptoms is present in an achondroplastic.

The bone lesion in cretinism is underdevelopment, as may be well demonstrated by means of the x-ray. Another point of differentiation lies in the fact that cretins recover under thyroid extract when treated early, whereas it has no effect whatsoever on achondroplastics. Umbilical hernia is the rule in cretins, but absent in achondroplastics.

Congenital Syphilis.

Achondroplasia may be mistaken for congenital syphilis. In syphilis the "pug nose" is due to actual bone disease; in achondroplasia it is due to a premature union of the bones at the base of the skull.

Osteogenesis imperfecta.

An x-ray diagnosis can readily be made at any age, except possibly in infancy, before ossification is normally far advanced at the knee, upper end of the femur and both ends of the humerus. It is most easily made during childhood, when these epiphyses are well developed; but again it requires more careful observation in adults, after these epiphyses have united and there are to be found only the results of the abnormal processes of development in these regions. At any age, however, separate and distinctly typical features are to be observed in the appearance of the shafts of the long and the short bones and also in the cancellous structure.

Comparison of Radiographic Appearances

The more important characteristic radiographic appearances can be classified conveniently in three groups as follows:[1]

Achondroplasia

(*a*) *Epiphyses of the Long Bones*

1. A moderate delay in the beginning of ossification.
2. A moderate delay in the process of ossification which tends later to an actual deficiency in development rather than to delayed union.
3. Although actually deficient in development, the epiphyseal ends in children and fully ossified ends in adults are relatively far better developed than the shafts of the bones.
4. Deficient and irregular ossification of the ends of the diaphyses of many of

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[1] From Pancoast's description in Fussell, Pancoast and McComb's article.

the bones is very evident, especially in early childhood or infancy, and particularly at the knee.

5. The bones of the carpus and tarsus and the patella exhibit a corresponding delay in ossification and resulting deficiency in development.

6. The long bones appear to manifest a relatively greater degree of development in the immediate neighborhood of epiphyseal centers, whether their growth is largely dependent on the latter, as at the knee, or is entirely independent, as at the olecranon and lesser trochanter. In the latter instance this is manifest even before epiphyseal ossification begins.

(*b*) *Shafts of the Long Bones*

1. These are much shorter than in normal individuals of the same age. This is characteristic of all of the long and the short long bones of the extremities.

2. The same bones are relatively thick for their length.

3. There is a tendency toward the growth of exostoses from the long bones of the upper extremities, with rather symmetrical arrangement on the two sides, but such growths are noticeably absent in the lower extremities.

4. There is decided bowing of many of the long bones, especially the tibia, femur, and those of the forearm. In the two first mentioned this is most marked near or at the epiphyseal ends, and is there shown to be largely a result of deficient and irregular development and ossification.

5. All of the bones and short long bones of the extremities present an abrupt expansion at the epiphyseal ends of the diaphyses, and to a width corresponding to that of the epiphysis.

(*c*) *Structure of the Bones*

1. There is a noticeable deficiency in development of the cancellous structure at the ends or epiphyses of the long bones and in those of the carpus and tarsus. It may be observed to best advantage, perhaps, in the os calcis. The appearance is characterized by relatively few walls and resulting large haversian spaces. The walls are usually more noticeable or better developed in one general direction. The appearance is quite different from that of the rarefaction so frequently seen in such conditions as chronic arthritis, and in which the walls have been absorbed and rendered thin and the spaces correspondingly large without any reduction in the number of the latter.

2. The medullary canals of the relatively thick bones are apt to appear too wide and the compact walls too thin for the diameter of the bone.

Cretinism

The x-ray diagnosis of this condition is by no means as easy or as certain as is the case in connection with achondroplasia, for the reason that there are fewer striking peculiarities and characteristic features to distinguish it from other conditions aside from achondroplasia. A similar and comparative summary of the case of cretinism follows:

(*a*) *Epiphyses of the Long Bones*

An exact comparison is difficult because of the difference in ages of this one patient and the other three under consideration, and the normal individual has been used as the basis of comparative data in this condition. The epiphyses appear to be generally deficient in size and development, and ossification is correspondingly delayed.

There is very noticeable delay in ossification of the carpal bones. There is not the relatively greater degree of development in the immediate regions of epiphyseal centers which is so noticeable in achondroplasia, and the ends of the diaphyses do not expand abruptly.

(b) *Shafts of the Long Bones*

The shafts are deficient in length actually, but not so relatively short as in achondroplasia. Moreover, the diameter or thickness is about right for the length. Exostoses are not present. Bowing is not a feature of importance in this condition.

(c) *Structure of the Long Bones*

The deficiency in the development of the cancellous structure so characteristic of achondroplasia is not a feature of this condition, in which this structure is not distinctly abnormal in appearance.

RICKETS

The radiograph of the lower extremities in a typical case of rickets and showing the characteristic appearance and deformities has been employed for the purpose of comparison.

(a) *Epiphyses of the Long Bones*

1. The extent of the ossification does not indicate a delay in the process for the age of the individual, although the amount of calcification may be deficient.
2. Ossification and development of the epiphyses are far more extensive than in the extreme ends of the diaphyses where the greatest resulting deformities exist, although calcification is deficient.
3. The ends of the ossified portions of the diaphyses are wider than the ossified centers in the epiphyses, indicating a wide zone of cartilage at the epiphyseal lines, which is typical of rickets.
4. The ends of the diaphyses show a decidedly irregular line of ossification resulting from temporary absorption of trabeculae and failure of ossification, but the appearance is quite different from that seen in achondroplasia at the same age.
5. The carpal and tarsal bones and patella do not share in the process to any extent and do not inhibit any delay in ossification or deficiency in development.
6. There is no apparent relative overdevelopment in the region of epiphyseal centers as in achondroplasia, especially in such regions as the trochanters.

(b) *Shafts of the Long Bones*

1. The long bones of the lower extremities are not actually so much shorter than normal, as in achondroplasia, though they may appear so because of resulting deformities. The short long bones are not affected noticeably.

2. Their thickness is not distinctly abnormal for their length.
3. There is no special tendency toward the growth of the exostoses from the bones of the upper extremities.
4. Bowing is common in both conditions and in itself is of no value in diagnosis. In achondroplasia it is largely developmental, while in reality it is quite different. The ends of the diaphyses are not ossified, or the ossification is very irregular at the exact locality in which the abrupt expansion occurs in achondroplasia (Fig. 111).

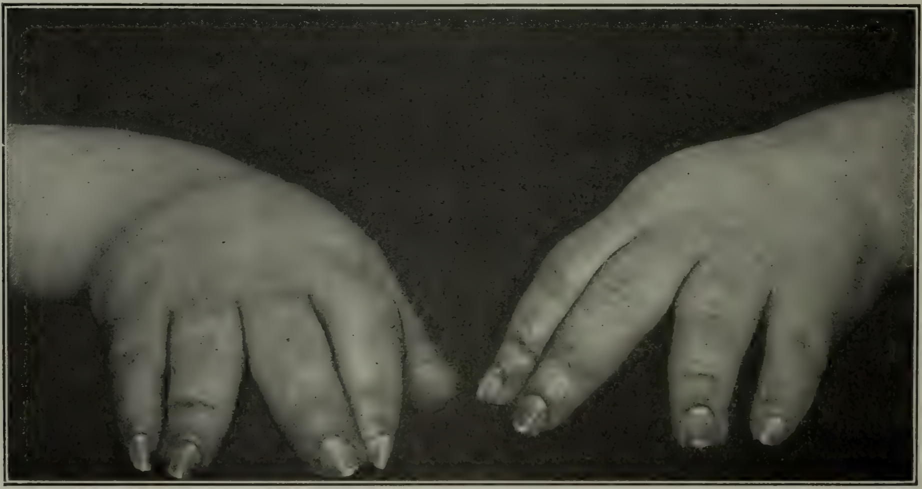

Fig. 111.—Trident Hands of Achondroplastic. (Original Observation.)

(c) *Structure of the Bones*

The trabeculae of the cancellous structure may become thinner or entirely disappear in rickets, but when visible, the peculiar appearance seen in achondroplasia is not evident.

5. Osteopsathyrosis

Osteopsathyrosis is a condition characterized by great brittleness of bones and consequent frequent fractures. It is further characterized by the fact that the general health of the patient is otherwise good though the fractures may number as many as a hundred or more in a single case. The fractures are generally painless and heal rapidly; they cannot be said to be spontaneous because they result from trifling causes, such as the mere turning over in bed, a slight blow, or even from so trifling a cause as chewing; the latter causing a fracture of the jaw. It contrasts further with fractures of the more usual kind in that it occurs in the young rather than the old.

6. Osteogenesis imperfecta

A condition of the fetus in which its bones fail to develop normally, reaching at birth a stage of great fragility wherein fractures are so easily produced that they may have occurred *in utero.* The defective development extends to the cranium and the fragility to all the bones. At other times the extremities are bent and deformed. Though the disease is commonly fatal the bones sometimes repair, and as the child grows older a natural firmness is acquired.

It can be mistaken for no other condition.

7. Oxycephaly

Oxycephalis is a deformity of the cranial vault resulting in abnormal vertical dimension associated with exophthalmos and defective vision without mental derangement. It is further characterized by feebly marked supra-orbital ridges. The forehead slopes to a pointed vertex and the scalp, rising abnormally, gives the appearance of being set on the top of a comb. Usually present at birth, it may occur as late as the sixth year.

The deformity is ascribed to premature synostosis of certain sutures —especially the coronal. The brain thus restricted grows vertically instead of laterally and anteroposteriorly. The closure of the anterior fontanelle is delayed and its site displaced, but closure ultimately takes place, the original site being covered by thin and prominent bone.

The visional defect is due to optic neuritis and atrophy caused by the internal brain pressure, as in tumor of the brain, whence too the exophthalmos and headache may be traced, and perhaps the occasional loss of the sense of smell.

Diagnosis.—*Oxycephaly cannot be mistaken for any other condition.*

Section XII

Diseases of the Nervous System

BY

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A. General Considerations

The diagnosis of most organic diseases of the nervous system is not more difficult than that of the affections of other organs if one possesses some knowledge of the anatomy and physiology of the brain, cord, and peripheral nerves.

For this reason it is thought advisable to precede the discussion of the various diseases with a very brief description of the *course and function of the important centers and tracts of the brain and spinal cord.*

B. Anatomy and Physiology of Nervous System

The nervous system is made up of units, termed *neurons.* Each neuron consists of a *cell body,* containing a *nucleus,* which in turn contains a *nucleolus,* one or more processes arising from the cell body and of similar structure, called *dendrites* or *dendrons,* and a single process of different structure which arises either from the cell body or a dendrite, termed the *axon* or *axis cylinder process.*

Cell Body.—The cell body, according to its location, may originate efferent impulses (cortical cells of motor area), modify impulses received from another neuron (ganglion cells in the anterior horns of the cord), or receive and recognize afferent or sensory impressions (sensory centers).

Certain cells, in addition, exercise a trophic or nutritive influence over muscles, bones, skin, etc. Thus the cells in the anterior horns of the spinal cord influence the nutrition of the muscles.

Axon.—Efferent impulses are carried away from the cell body by the axon, which may end either by surrounding the dendrites of another neuron with its end brushes, or if from the peripheral motor neuron, end in the motorial end plates in the muscles (Fig. 114). In the former cases they would form the so-called motor tract as they pass through the brain and cord, in the latter they would form the motor fibers of the peripheral nerves. Each axon is surrounded by a myelin sheath.

Dendrites.—The dendrites convey afferent or sensory impulses and nutriment to the parent cell. The sensory portion of the peripheral nerves is composed of dendrites (p. 556). The white matter of the nervous system consists of axons, the gray matter, of collections of cell bodies.

Centers.—A group of cells which together control some function of the body is called a center. Thus the group of cells located in the upper portion of the precentral convolution, which controls the movements of the leg muscles, is known as the *leg center.*

Cortical centers are both motor and sensory, and are shown in Figures 135 and 136.

The various so-called *tracts* of the brain and cord are composed of white matter, and hence of axons. These also constitute the motor fibers of the peripheral nerves. Those fibers which connect cortical centers directly with either the cells of the basal ganglia, the nuclei in the pons and medulla, or gray matter of the spinal cord, are termed *projection fibers.*

Other groups of cells not connected with projection fibers act as stations where the various sensory impressions are collected, arranged and coördinated. These are termed *association centers.* The fibers connecting these with motor, sensory and special sense centers are known as *association tracts.* Fibers which connect the two sides of the brain are called *commissural tracts or fibers.*

The projection fibers form the pathways by which the motor impulses are carried from the cortical centers to the cells in the anterior horns of the cord and by which sensory impressions are brought from certain ganglionic masses in the medulla and base of the brain to the cerebral cortex.

Tracts.—These pathways, together with certain columns in the spinal cord and the peripheral nerves, constitute the *motor* and *sensory tracts.* It will be noticed, therefore, that these tracts are composed of two or more neurons.

There are other tracts which probably have to do with regulating motor impulses and muscle tone by connections with the autonomic and sympathetic or vegetative nervous system—vasomotor, secretory, and respiratory functions, emotional expression, and movements of involuntary muscles.

Motor Tracts.—The motor tracts are known as the *pyramidal tracts,* each of which is composed of two neurons—the upper or central which runs from the cortical cells in the motor centers to the cells either of the nuclei of the motor cranial nerves or those in the anterior horns of the gray matter of the cord, and the lower or peripheral which runs from these cells to the muscles (Fig. 114).

Those having to do with muscle tone, etc., are called the *extrapyramidal tracts.* These have connections with the corpus striatum, cerebellum, optic thalamus and red nucleus. Their cortical centers are probably in the parietal lobe.

Sensory Tracts.—The course of sensory impulses from the periphery is not so well known as the motor.

There are different pathways for the various forms of sensation, viz.: tactile, pain, temperature, muscle and pressure. The cells of the first neuron are situated in the ganglia upon the posterior nerve roots, or sensory cranial nerves, as the case may be. Each cell gives off a long dendrite which runs to the periphery and ends in one of the various end organs in either the skin, muscles, joints or organs of special sense, according to the particular form of sensation which it conducts.

The axon enters the spinal cord, if a spinal nerve, or the cerebrum, medulla or pons, if a cranial nerve. In the case of spinal nerves it divides after entering the cord into a long ascending and a short descending branch. Many of the former pass up the cord in the posterior columns (Goll and Burdach) and end in the nucleus gracilis and nucleus cuneatus respectively. The cells of these nuclei are the beginning of another neuron, the axons of which form the lemniscus or fillet. This also receives fibers from the sensory cranial nerve nuclei. Here the fibers decussate and go to cells in the optic thalamus of the opposite side. These cells form the beginning of another neuron, the axons of which pass through the posterior part of the posterior limb of the internal capsule to the parietal region of the cortex (Fig. 135). *Tactile sensation* is largely conducted by this path.

Sensations of pain and temperature are conducted to the cortex by *Gowers' tract* and also the *tractus spinothalamicus et spinotectalis.*

The tracts are formed by fibers running to the cells in the posterior horns of the gray matter of the cord. From these cells axons arise which pass to the opposite side and form the tracts above mentioned, in the anterolateral region. The cranial sensory nerves (fifth, ninth and tenth) also join these tracts in the pons and medulla.

Sensations from muscles and joints probably reach the brain by the direct cerebellar tracts, which are formed by axons from the cells of Clarke's column and by fibers which pass upwards through the posterior columns to the nuclei gracilis and cuneatus. From these, fibers, together with those of the direct cerebellar tracts, pass by means of the inferior cerebellar peduncles to the cerebellum, hence to the optic thalamus and red nucleus and hence to the cortex. These tracts have to do with muscle sense and coördination.

The sympathetic system is connected with cells of the intermediolateral tracts and "lateral horn group."

C. General Symptomatology and Methods of Examination

The general term for any derangement of the nervous system, except those of the mental functions, is *neurosis.* Usually, however, this term is applied to the so-called functional disorders or those in which no organic lesion can be found, as neurasthenia. Disturbance of the higher or mental functions, as memory, judgment, etc., is termed a *psychosis.*

The symptoms caused when either a nerve center or tract is diseased depend upon whether the lesion is irritative or destructive and also if it exerts pressure upon neighboring tracts and centers. An *irritative lesion* causes increase of function; thus if the moter centers are irritated there is at times increased action, manifested by Jacksonian convulsions (p. 614). A *destructive lesion* causes diminution or loss of function; thus if the motor cortical centers are destroyed, paralysis of the parts which they supply results.

A lesion may at first be irritative and later become destructive; thus a tumor springing from the meninges may at first irritate the motor cortical centers, causing convulsions or spasm, and later may by its increased growth destroy them with resulting paralysis. A lesion may also destroy one part and irritate neighboring parts or it may by pressure also interfere with the function of surrounding tracts and centers. A lesion of one center dependent upon or related more or less in its function with another center located some distance away, may interfere with the nutrition and function of that center. Thus destroyed function of one speech center interferes more or less with the action of the other speech centers (p. 616).

Symptoms resembling those of irritation may be caused by interference with the function of the inhibitory apparatus. Thus if the lenticular nucleus which has something to do with regulating muscle tone is destroyed, there is overaction of the motor tract, as shown by spasticity, tremor and contractures (p. 688).

1. Symptoms Caused by Increased Action of the Motor Tracts

(*a*) *Convulsions*

One of the most common of these is a convulsion. This has been defined to be "a violent involuntary contraction or series of contractions of the voluntary muscles" (Dorland). They depend either upon irritation of the motor cortical centers by either an organic lesion, poison, or an excessive paroxysmal discharge of energy generated in an unknown way, or the action of certain poisons (strychnin, tetanus toxin) on the spinal cord. Consciousness may be either preserved, as in spinal convulsions and Jacksonian epilepsy (p. 614), or lost. If the latter, they are termed *epileptiform.*

Spasms.—Convulsions are also termed *spasms,* but this term is more commonly applied to those of limited or local distribution. They are also divided into *tonic* and *clonic.* In the former case the contraction is slow and continuous, in the latter the muscles rapidly and alternately contract and relax.

When the muscles are in a long continuous state of hypertonicity or overcontraction, they are said to be *spastic.* If this is confined to a group of muscles which overpower their antagonists and cause a deformity, it is termed a *contracture.* For example, the overflexion of the hand and the forearm seen in most cases of hemiplegia following apoplexy. If actual shortening of these muscles occurs, as usually is the case in time, it is known as a *contraction.*

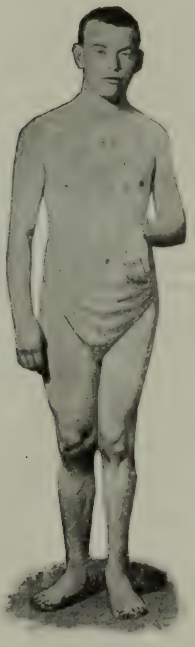

Fig. 112. — Spastic Hemiplegia with Epilepsy. (Philadelphia Hospital.)

Spasticity is caused by an increase of muscle tone. This is probably exerted through the extra-pyramidal tracts (p. 556) controlled by an inhibitory center or centers, the exact location of which is not known but is partly at least located in the lenticular and red nuclei.[1] All of which has been termed by Mills the "tonectic apparatus," the cortical centers for which he believes to be in the frontal lobes anterior to the motor centers.

If the action of the governing centers is interfered with and the tract is active, more or less hypertonicity results. If, however, the extrapyramidal tract is interfered with, more or less hypotonicity results. Contractures may also be functional, as seen in hysteria.

These conditions can be discovered by passively flexing and extending the limb, when abnormal resistance will be noted. Deformity due to the overpowering of the extensors by the flexors is also often present. It should be borne in mind that in those of long standing actual ankylosis may occur (Fig. 112).

Functional contractures, unless of very long duration, can be distinguished from those of organic cause by the fact that they disappear during sleep and under the influence of an anesthetic.

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[1] It has until recently been thought that inhibition or control was exerted through the pyramidal tracts. Late studies seem to disprove this.

In this connection attention must be called to a symptom termed by Liepmann *"tonic perseveration"* and by Wilson (Brain, 1914, p. 199) *"tonic innervation."* It consists of inability, owing to a central lesion, to relax a given innervation in any muscular group or groups; thus if a patient with this symptom grasps an object he is unable to let go of it. The condition must not be confounded with myotonia (pp. 530-531) in which there is no central lesion, so far as is known. It is due to inability to inhibit the contraction of certain groups of muscles after it is once begun.

The symptom has been present in patients who had a lesion in the midfrontal region (where Mills has placed his centers for muscle tone) (p. 558) on the side opposite to that in which the phenomenon occurred. Weakness, not marked, of the affected side is also present with symptoms of disturbance of the pyramidal tract.

The term *perseveration* has also been applied to two other symptoms, viz.: "clonic perseveration" and "inten-

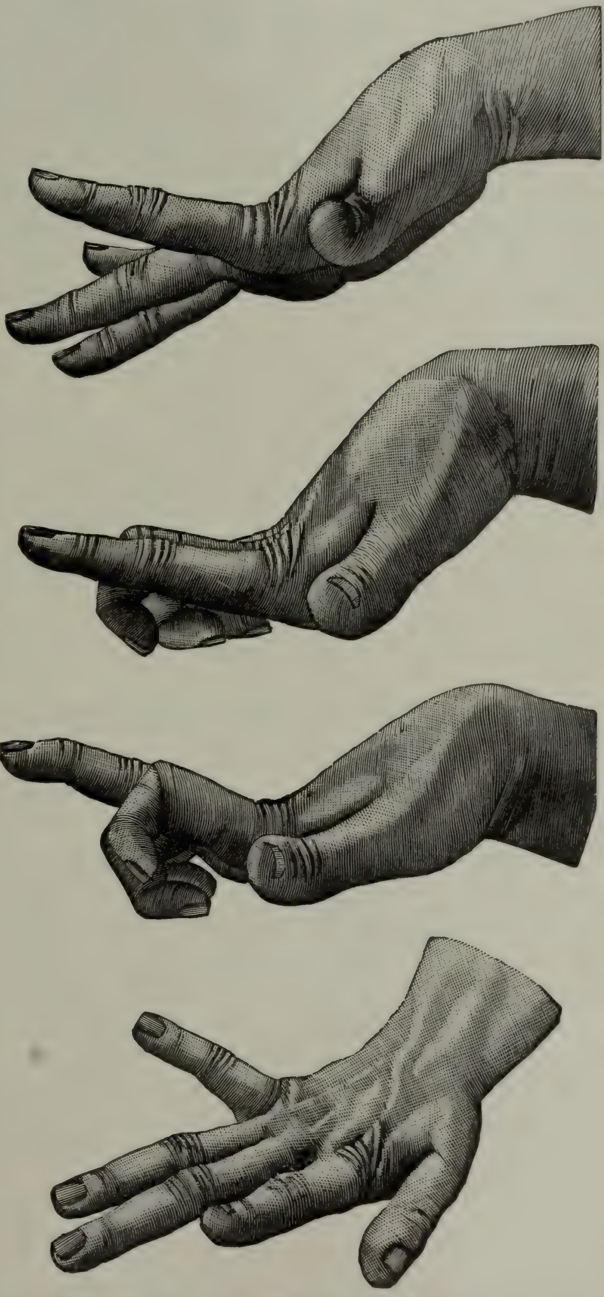

Fig. 113.—Example of the Position of the Fingers in the Movement of Athetosis. (After Strümpell.)

tional perseveration." These phenomena are different from that just described (p. 617).

(*b*) *Athetosis or Athetoid Movements*

Slow, irregular, vermicular, involuntary movements of the fingers, toes, and sometimes of the face have been so termed (Fig. 113). They are usually increased by excitement or muscular effort, and are most frequently seen associated with the cerebral palsies of childhood (p. 689).

They are probably due to lesions in the red nucleus and vicinity which, as has been said, probably acts as a control of muscle tone (p. 558).

True athetosis from similar lesions rarely occurs in adults; intermittent tonic spasms, known as *hemihypertonia postapoplectica,* are more common, as are also *tremor* and *choreiform movements.*

(*c*) *Tremor*

By tremor is meant a to-and-fro movement of a part due to the involuntary, rhythmic and alternate contraction of antagonistic muscles. It may be either rapid or slow, fine or coarse, and the limbs, neck, face or tongue may be the seat. It is probably most common in the arms.

Tremor is a phenomenon of unstable or irregular tonicity (Mills). It may be either only present during voluntary movement (intention tremor), constant but increased by voluntary effort, or constant but ceasing or diminishing for a short time during voluntary movement of the affected part.

Nystagmus (p. 583) is a form of tremor of the eyeballs.

Tremor is a symptom due to many different causes and found in many different conditions. The table on the opposite page gives the various causes and the character of the tremor present.

Examination.—In studying a patient with tremor it must be noted whether it is fine or coarse, intentional, constant, increased by movement of the affected parts or diminished by it. If a tremor is not apparent it may be discovered either by making the patient extend the arms and hands at full length with the fingers separated, especially if a sheet of paper is laid across them, or by causing him to perform some voluntary act, as drinking a glass of water.

An intention tremor may be discovered by having the patient endeavor to touch the end of his nose with his finger (finger to nose test).

Tremor of the tongue can be discovered by causing it to be protruded. It may be more or less fibrillary (p. 561) in type, or the tongue may go in and out like the piston of an engine (the so-called piston or trombone tongue seen in paresis).

Causing the patient to show the teeth or whistle will bring out *tremor of the facial muscles.*

Cause	Type of Tremor	Rapidity
Toxic: Alcohol Lead Mercury Tobacco Tea Coffee Arsenic Exophthalmic goiter	Intention in early stages; later may become constant, then usually increased by voluntary movement	Rapid except mercury and alcohol which may be slow
Neuroses: Hysteria Neurasthenia	That of hysteria may simulate any other form of tremor. In neurasthenia it is usually seen best during muscular effort	May be slow or rapid; usually rapid
Senility. Arteriosclerosis	Usually constant; increased by exertion; may be intentional at first	Slow
Any disease of brain or cord which interferes with "tonectic apparatus" (p. 558), excepting multiple sclerosis and paralysis agitans. More or less frequently follows apoplexy	Ibid.	Slow
Degeneration of certain parts of cerebellum Dyssynergia cerebellaris progressiva of Hunt Pseudosclerosis Lenticular degeneration (Wilson's disease) (p. 688)	Intention at first; later constant; increased by volitional movements	Slow
Paralysis agitans	Often ceases for a few seconds during and after muscular exertion	Slow
Multiple sclerosis	Intention only	Fine at first, becoming slow; somewhat irregular
Cerebrospinal syphilis	Intention or constant	Usually slow
Paresis	Ibid.	Either slow or rapid
Hereditary	More or less constant; worse during effort	Rapid

Excepting that of paralysis agitans all tremors tend to become more marked during movement of the affected parts. Those due to organic disease are usually coarse, those of toxic origin are usually fine.

(*d*) *Contraction*

Fibrillar or Fascicular Contraction.—A fibrillar or fascicular contraction is an involuntary contraction of small numbers of muscle fibers, not as a rule dependent on movement. It may consist either of quivering of the muscle or of wavelike contractions running along the muscle in which they occur (myokymia). Movement of the part is not caused by them although they may sometimes be caused by movement. Tapping or other mechanical irritation of the muscle will also excite them.

They are most frequently seen in muscles that are degenerating from loss of neurotrophic influence, as in progressive spinal muscular atrophy, amyotrophic lateral sclerosis, syringomyelia, bulbar palsy and chronic anterior poliomyelitis. They are sometimes seen in exhausting diseases, neurasthenia, neuritis, arthritic atrophy, and after exposure to either cold or excessive heat.

Myoidema.—A condition known as myoidema may also occur in such muscles. It consists of the formation of a ridge of nodules when the muscle is squeezed or a hard substance drawn across it. It is most frequently seen in the biceps and pectoral muscles.

(*e*) *Choreiform Movements*

Choreiform movements are sudden, incoördinate, non-rhythmical, non-purposive movements of different groups of muscles. They usually cease or diminish during voluntary effort and during sleep. They must be distinguished from the movements of tic (p. 764).

(*f*) *Forced Movements*

Forced movements are those in which the patient is compelled, against his will, to move in a certain direction, as to one side, forward or backward, or to rotate. They are usually seen in cerebellar disease and in the gait of paralysis agitans.

(*g*) *Associated Movements*

If moving a non-paralyzed limb causes movement in a paralyzed one, the latter is termed an associated movement.

2. Symptoms Due to Destructive Lesions of the Motor Tract

When any part of the motor tract is the seat of a destructive lesion, either loss or impairment of function of certain muscles occurs, depending in extent upon the seat and severity of the lesion. This is known as *motor paralysis*. If the function is only diminished it is sometimes termed *paresis*. When one limb is affected it is termed *monoplegia*. When all or most of the muscles of one side of the body are involved we call it *hemiplegia*. Paralysis of like parts on each side, as both arms, is termed *diplegia.* This term is most commonly applied to involvement of both legs and both arms. If both legs only are affected, the term *paraplegia* is employed. When the cranial nerves of one side and the arm and leg of the other are affected we speak of *alternate or crossed paralysis.* If abnormal muscular rigidity is present the paralysis is said to be *spastic paralysis.*

If the lesion is in the pyramidal tract, i. e., either in the cells of the motor area of the cortex or in the axons leading from these cells to the ventral horn cells in the cord, the paralysis is said to be *central* or of the central or upper motor neuron. If it involves either the nuclei of the motor cranial nerves, cells in the anterior horns of the cord or in the axons leading from them to the muscles, the paralysis is said to be *peripheral* or of lower motor neuron (Fig. 114).

The symptoms of either central or peripheral paralysis are characteristic and are noted in the following table:

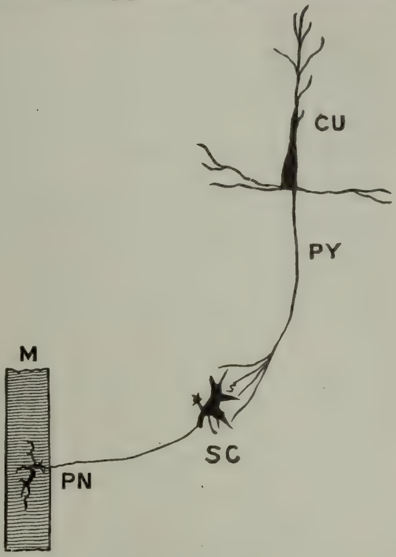

Fig. 114.—CU Represents a Cell in the Motor Region of the Brain Cortex; PY Is Its Axone, Which Forms Part of the Pyramidal Tract; SC Represents a Cell in the Gray Matter of the Cord (Anterior Horns); PN, Its Axone, Forming Part of a Peripheral Nerve; M, Muscle. A Lesion Destroying CU or Any Part of the Tract PY Causes a Central Palsy; a Lesion Destroying SC or Any Part of the Tract PN, a Peripheral Palsy. (After Potts, "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

TABLE SHOWING THE DIFFERENCE BETWEEN CENTRAL AND PERIPHERAL PARALYSES

	Diseases of Upper or Central Neuron	Diseases of Lower or Peripheral Neuron
Nutrition of Muscles	Good. There may be apparent atrophy from disuse and in such lesions occurring in early childhood, but it is not real atrophy, merely lack of growth	Poor. Muscles more or less atrophied
Tone of Muscles	Increased. Muscles usually more or less spastic unless extrapyramidal tract is destroyed	Diminished. Muscles more or less flaccid
Reflexes (p. 587)	Tendon jerks increased unless extrapyramidal tract is destroyed. Babinski present if leg is involved	Diminished or lost. Babinski absent
Electrical Reactions (p. 603)	Same as normal muscle	Changed. Either quantitative decrease or reactions of degeneration

A lesion of a peripheral nerve involves the muscles supplied by that nerve; if in the cranial nerve nuclei or anterior horns of the cord the muscles supplied from the particular cells involved are paralyzed. A lesion in the cortical motor centers causes paralysis of the muscles supplied from the center involved and also in some cases, if the lesion makes pressure, of those supplied from the neighboring centers. If the pyramidal tract is cut off anywhere in its course all muscles supplied by nerves arising from the pons, medulla or cord below the seat of the lesion are involved.

Methods of Examination.—In examining a case of suspected motor paralysis it must be remembered that muscular weakness may be due to general exhaustion from acute illness or rachitis, that the movements of a limb may be restricted either by a complete or partial joint ankylosis or by pain produced by movement. It must also be borne in mind that paralysis may be due to disease of the muscles, either inflammatory or degenerative (pp. 523 and 536).

In many cases the existence of paralysis is self-evident. The inability to use the part, if an entire limb is affected, or the resulting deformity and inability to make the characteristic movement when a group of muscles is involved at once tells the story; for instance, the wrist drop when the extensors of the hand are affected or drooping of the eyelid when the elevator of the lid is involved.

When the paralysis is not complete, some power in the muscles remaining, certain tests are useful. In this connection it must be remembered that in making every muscular movement muscles are used in addition to those which appear to actually do the work. Thus in using the flexors of the hand and fingers in grasping an object tightly the extensors must also contract, otherwise the flexor muscles would cause flexion of the hand and the grasp be weakened. The extensors in such a case are known as *fixation muscles*, and prevent a movement being made that is not desired.

In making certain movements apparently non-active muscles may be brought into play in another way; thus the biceps is the principal supinator of the forearm, it also flexes the forearm on the arm. When supination is the movement desired the triceps also contracts to prevent flexion of the forearm. Muscles acting in this way are known as *synergic muscles.*

In making any movement the antagonistic muscles must relax; thus in extending the forearm on the arm the biceps, brachialis anticus and supinator longus, which are antagonistic, i. e., muscles acting in the opposite direction to the biceps, must relax. As will be seen further on, this may be used in differentiating functional from organic paralysis.

In making some movements more than one muscle may be used under certain conditions; thus in flexion of the forearm on the arm the biceps, brachialis anticus and supinator longus are used, but if only a slight effort is required only the biceps contracts, but if a heavy resistance is being overcome all of these muscles contract.

In examining a patient *to detect weakness,* we ask the patient to use the suspected muscles, comparing the movements with those of the opposite side. In very mild cases the movement may appear normal when made once or twice but become weak from early exhaustion if made several times. In the case of the limbs these movements may be made against the resistance of the examiner. Thus, in testing flexion of the forearm, the examiner grasps the wrist and endeavors to prevent the patient from making the movement, doing this first on the one side and then on the other and comparing the amount of effort necessary to prevent the movement in each case.

An instrument known as a hand dynamometer may be utilized for testing and recording the *relative strength of the hand grasp.* This is used by holding the instrument in the hand, squeezing it as hard as possible, and noting the figures to which the indicator points.

In examining the *muscles of the face* cause the patient to open and close the eyes, wrinkle the forehead, smile, whistle, show the teeth and draw the angle of the mouth from side to side. Weakness of the *muscles of mastication* may be detected by placing the fingers over the masseter and temporal muscles of each side and asking the patient to bring the jaws firmly together. Difference in the amount or absence of contraction will be noted.

The *external pterygoids* are tested by causing the patient to move the lower jaw from side to side. Weakness is shown by inability to move it toward the normal side and by deviations of the point of the chin towards the weak side when the mouth is opened widely. Weakness of the *internal pterygoids* causes inability to push the lower jaw forward.

Paralysis of the *soft palate* is discovered by asking the patient to open the mouth and make the sound "Ah." If both sides are involved the palate will move but slightly or not at all. If only one side is involved it will be drawn up on the sound side only.

Weakness of the *tongue* is discovered by noticing if it is protruded with difficulty or not at all, in which case the muscles of both sides are affected, or if when protruded the tip goes to one side, the muscles of the side toward which the tongue goes are weak. If the weakness is slight the tongue may be protruded but cannot be kept so long.

In examining the *muscles of the eyeball,* we cover one eye and ask the patient to move the eye in different directions, the head being kept fixed. Associated movements upward and laterally should also be tested. Slight weakness may be only shown by diplopia or double vision. This is usually complained of when there is weakness of any of these muscles. In doubtful cases an ophthalmologist should be consulted.

To test the *movements of the iris,* the eye not being tested is covered.

A bright light is brought before the eye when, if normal, it should contract and dilate when it is removed. The light should be brought into the field from the side to prevent the influence of accommodation as the iris may be paralyzed when the eye is stimulated by light, but not when the effort to accommodate or converge is made, and vice versa (p. 592). To test the latter the eye is made to look first at a far object than at a near one. The finger is usually used as the object looked at. Sometimes after the pupil contracts it will dilate and then contract again while exposed to the stimulus; this may happen a number of times. This phenomenon is known as *hippus.* In other cases after contraction it dilates and remains so even while exposed to the light. This is known as a *rebounding pupil.* Both these phenomena may be evidences of weakness of the iris.

The *limbs* are tested by causing the patient to make the various movements, comparing one side with the other or against the examiner's resistance as described above. If the patient is comatose, paralysis of the limbs may be detected by noticing that if the limb is raised and allowed to drop there is less resistance on the paralyzed than on the sound side.

The presence or absence of muscular flaccidity and atrophy must be noted. In this connection it must be remembered that even in central nervous lesions (p. 563) there may be slight atrophy which is due to disuse. When such lesions occur in early childhood (p. 689) there is frequently lack of development of the entire affected limbs, not only muscles but bones also. In such a case it will be noted that the muscles while small are firm and will respond normally to the electric current (p. 610). If slight the limbs must be measured. To do this we select a fixed point, as the interior iliac spine, from which at equal distances on each side we make a number of points. At these points we measure the circumference of the limbs and compare results. Of course allowance must be made for the normal differences between the right and left sides.

Electrical tests (p. 603) should also be made. It may be necessary to distinguish between paralysis due to an organic lesion and that due to functional disturbances, as in hysteria. The means of doing this are detailed on pages 604, 607, 609.

In studying paralysis of the legs attention to the *gait* is important. It must be remembered that we may have disordered gait without muscular weakness, as in tabes dorsalis. The different forms of pathological gait are described on pages 582, 586, 672, 686, 698, 699, 708, 734, 780, 785.

3. Symptoms Due to Irritative Lesions of the Tracts Conducting Sensations of Pain and Touch

Such lesions cause either *hyperesthesia, pain* or *paresthesia.*

(*a*) ***Hyperesthesia***

By the first we mean an increased sensitiveness of the skin or special senses so that an ordinary or possibly very mild stimulus causes either discomfort or pain. When painful sensations are caused we may also speak of it as *hyperalgesia.* It may be found in certain visceral disorders (p. 568), in hysteria as the so-called hysterogenous zones (p. 778), and due to irritation of the posterior nerve roots as in meningomyelitis (p. 628).

(*b*) ***Pain***

Pain may be either dull, sharp and shooting, constant, paroxysmal, limited in distribution or diffuse. Dull pains may be present in disease of the spinal cord and sometimes in neuritis. They may be mistaken for ordinary rheumatic or muscular pains. Sharp, shooting, paroxysmal pains are characteristic of neuralgia and also of irritation of the posterior nerve roots. They occur in meningitis, vertebral disease, tabes dorsalis, intrathoracic aneurism and tumor of the cord, and when due to such causes are termed "root pains."

The shooting pain of neuralgia differs from those due to organic causes in that there is apt to be constant pain in the intervals; if organic, there is also apt to be anesthesia in the painful area (anesthesia dolorosa). The so-called girdle pain or sensation of a tight band being about the body, often present in diseases of the cord, is due to irritation of the posterior roots. Pain in the back aggravated by jarring and relieved by stretching the back is due to vertebral disease of some nature. Rigidity of the back muscles will also be present. Backache, associated with tenderness on very light pressure, is characteristic of neurasthenia or hysteria. Pain in the chest extending into the arm may be due to angina pectoris or to hysteria. A characteristic pain of neuritis is a sensation of intense burning which has been termed "causalgia." If the affected part is near the surface, as an inflamed nerve, tenderness may also be present. Irritative lesions of the cortical centers may cause pain to be referred to the limbs; this is especially true of lesions in or near the optic thalamus.

When an irritation of one side of the body is felt at the corresponding point on the other it is termed "allochiria." It is a symptom found in hysteria. Allied to this, and due to the same cause, are *achiria,* in which, while the patient knows there is a stimulus, he cannot tell on which side it is, and *synchiria,* when it is referred to both sides. *Alloesthesia* or *false allochiria* is the term used when the point stimulated is located by the patient either on the same or opposite side, but at a widely different point than that actually touched. This symptom is found in organic disease.

Pain may be referred to remote parts if the nerve supply is the same, as the knee pain of hip joint disease. The observations of Head (Brain, 1893, p. 1; 1894, p. 339; 1896, p. 153; also Elsberg & Neuhof, Am. J.

Med. Sci., June, 1908) have shown that definite and constant areas of cutaneous hyperesthesia and pain may be due to disease of the different viscera (Figs. 115-120).

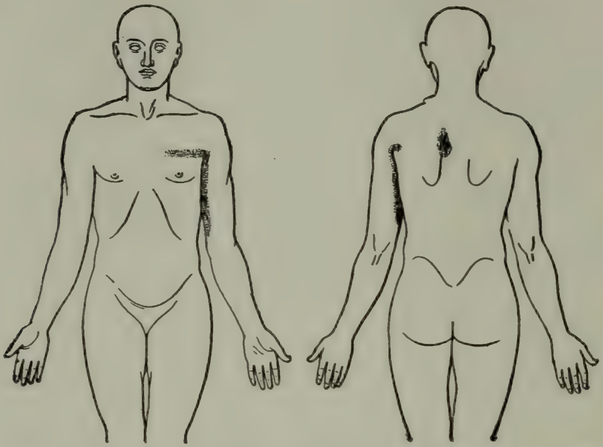

Fig. 115.—The Location of Reflex Heart Pains. (After Dana's "Textbook of Nervous Diseases," published by William Wood & Co., New York.)

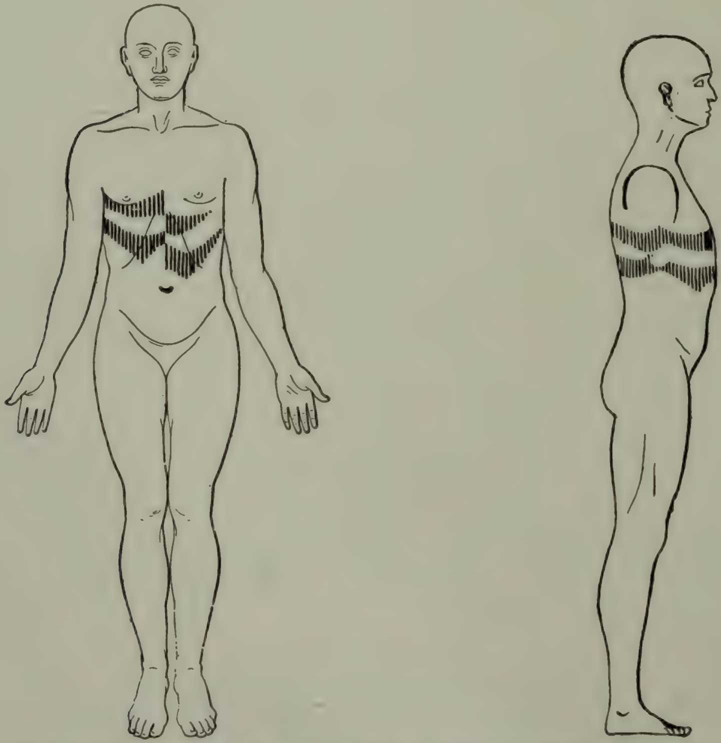

Fig. 116.—Areas of Cutaneous Hyperesthesia in Disease of the Stomach. Sixth, Seventh, Eighth and Ninth Dorsal Nerve-Segments. (After Head-Fraenkel.)

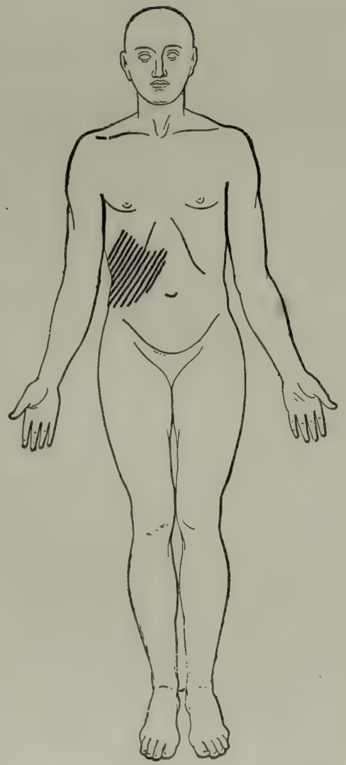

Fig. 117.—Area of Cutaneous Hyperesthesia in a Case of Cholelithiasis and Cholecystitis. (After J. Fraenkel.)

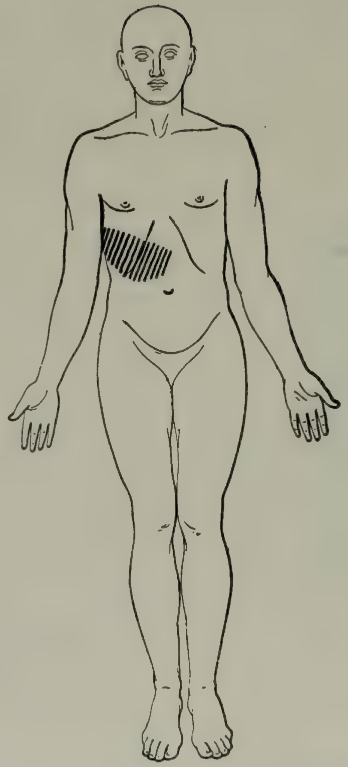

Fig. 118.—Area of Cutaneous Hyperesthesia in a Case of Cholecystitis. (After J. Fraenkel.)

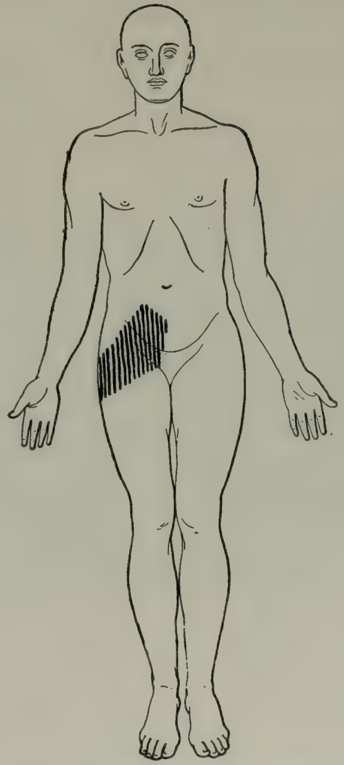

Fig. 119.—Area of Cutaneous Hyperesthesia in Appendicitis. (After J. Fraenkel.)

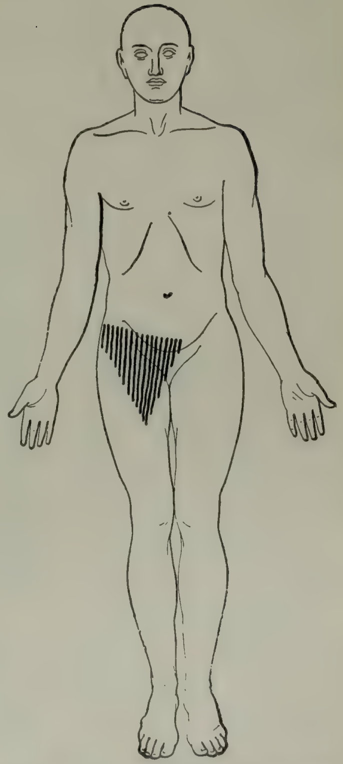

Fig. 120.—Area of Cutaneous Hyperesthesia in Case of Salpingitis. (After J. Fraenkel.)

Pain may follow the course of certain nerves and, as is often the case in some forms of neuritis, be absent or slight during rest but excited or increased when the afflicted part is moved.

Headache

Pain in the head, while often a symptom of disease of the nervous system, may also be a referred pain (Fig. 121). It is such a frequent symptom that it demands special mention. *Headache*, also termed *cephalalgia,* may be either paroxysmal or continuous, diffuse, or more or less localized, but it is not confined to the distribution of any particular nerve. The following table gives the different *causes:*

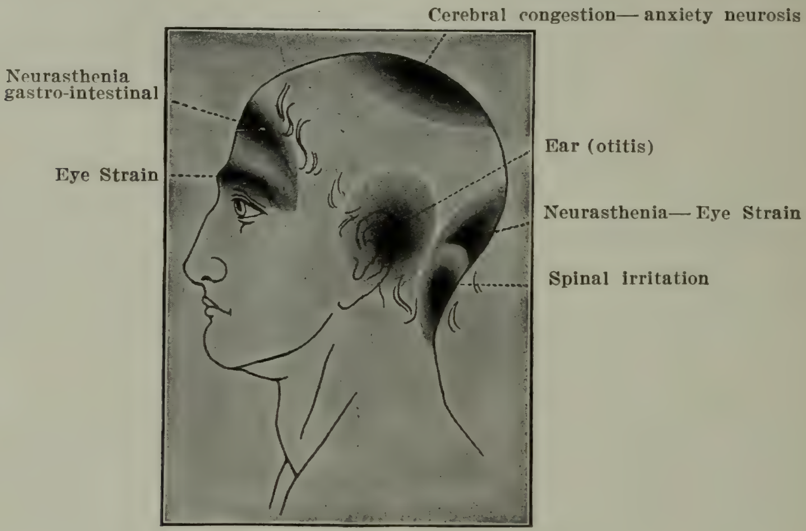
Cerebral congestion— anxiety neurosis
Neurasthenia gastro-intestinal
Eye Strain
Ear (otitis)
Neurasthenia— Eye Strain
Spinal irritation

Fig. 121.—Reflex and Symptomatic Head Pains. (After Dana's "Textbook of Nervous Diseases," published by William Wood & Co., New York.)

1. *Reflex Irritation.* Ocular (eye strain or inflammation); nasal and pharyngeal disease; disease of accessory sinuses; middle ear; decayed teeth or abscesses at the root; reproductive organs (especially female); disease of thoracic and abdominal viscera (Fig. 121).

2. *Toxemia.*

(*A*) *Infections,* as prodrome of acute infectious diseases; malaria; syphilis before secondary symptoms appear.

(*B*) *Metabolic and Defective Elimination,* as uremia, diabetes, gout, rheumatism, gastrohepatic derangements, constipation, exophthalmic goiter.

(*C*) *Action of Drugs and Poisons.*

(*a*) Acute, nitrites, quinin, opium, alcohol, carbon monoxid and dioxid, etc.

(*b*) Chronic, lead, tobacco, alcohol, opium, tea, coffee.

3. *Circulatory Disturbances.*

(*A*) *Passive Congestion,* as by posture; tight clothing about the neck; pressure on veins by tumors; disease of right side of the heart; emphysema and other diseases of the lungs which prevent the free circulation of the blood.

(*B*) *Active Hyperemia,* as early stage of acute meningitis; prolonged mental exertion.

(*C*) *Anemia* following loss of blood or the idiopathic anemias, as chlorosis; diseases of the heart which prevent a full supply of blood reaching the brain, as aortic stenosis, fatty heart, arteriosclerosis.

4. *Neuroses,* as epilepsy, hysteria, neurasthenia.

5. *Organic Disease of the Brain or Its Membranes:* viz., meningitis, syphilitic, traumatic or infectious; encephalitis; abscess; tumor; aneurism. (The brain is insensitive and pain in organic diseases of the brain is due to involvement of the meninges either actually or by pressure.)

6. *Caries of the Cranial Bones.*

7. *Indurative or Muscular,* due to the pressure of rheumatic nodules in the muscles of the scalp or neck.

8. *Migraine* (p. 772).

The nerve supply of the meninges accounts for the frequency of headache accompanying disturbances of other organs. The supply of the falx tentorium, and anterior three-fourths of the dura is derived from the fifth nerve; the remaining fourth from the sensory fibers of the pneumogastric. The scalp as far back as the vertex is also supplied by the fifth, the posterior portion being supplied by the posterior branches of the upper four cervical nerves.

The sensory or descending root of the fifth nerve is in close relation with the origin of the cervical nerves in the cord and also of the cranial nerves in the medulla. The regions of the dura, supplied respectively by the fifth and pneumogastric nerves, overlap. The viscera are also supplied in large part by the latter and in addition the pia receives branches from the third, fifth, sixth, seventh, ninth, tenth and eleventh cranial nerves.

In all cases of chronic headache a careful search must be made for the cause. According to this it frequently presents characteristics that may assist in the diagnosis. The pain may be pulsating or throbbing; hot, burning or sore; boring or sharp. In location it may be either frontal, occipital, parietal, temporal, vertical or diffused (Fig. 121).

A pulsating or throbbing pain, situated either in the vertex or diffused is frequently due to disturbance of the circulation; if burning and vertical, anemia should be thought of. A dull, heavy frontal pain is often due to a toxemia of some sort. The feeling as if a band were about the head or a feeling of pressure on top of the head is characteristic of neurasthenia. In such cases the pain is apt to be aggravated by exertion or excitement. The sharp, boring type is found in the hysterical and neurotic. It is often said to resemble a nail being driven into the head and is then known as *"clavus hystericus."*

Headache due to eye strain is aggravated or brought on by using the eyes and is located either in the occipital region or just over the orbit or possibly in both. Inflammatory conditions of the eye and glaucoma also cause headache; if due to the latter it is usually temporal. When due to inflammation of the nose and accessory sinuses, especially the frontal, the pain usually appears an hour or two after rising and improves towards evening; it is aggravated by lowering the head or jarring it in any way. The location is frontal but it may involve the entire head. There may be tenderness over the frontal sinuses. When due to syphilis the pain is sometimes only present at night and always much worse at that time. If the meninges are involved from any cause, there is apt to be tenderness if the head is percussed. This may also be the case if either an inflammatory or rheumatic condition exists in the scalp. Sometimes in cases of brain tumors there may be localized tenderness over the tumor.

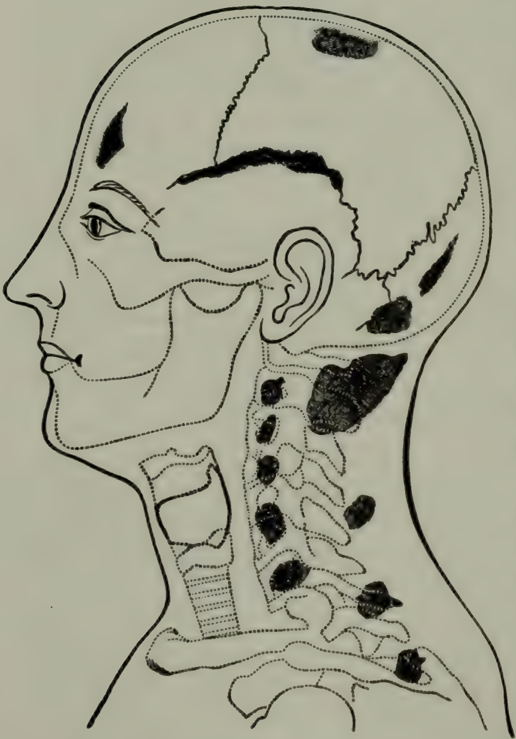

Fig. 122.—The Points Upon Which "Indurations" Are Most Frequently Found.

In indurative headaches a history will usually be gotten of either exposure to cold winds, washing the hair, or the rheumatic or gouty diathesis. The character of the pain closely resembles that of migraine (p. 772). Areas of tenderness, often with swelling and induration, will be found in the muscles of the neck and where they are inserted into the skull (Fig. 122). Edinger states that "examination of these muscles should never be neglected in any case of headache."

Accompanying Symptoms.—Vertigo, nausea and somnolence may be accompanying symptoms in any case of headache.

Differentiation.—In studying a case of head pain it must first be distinguished from NEURALGIA (*infra*). In this the pain is shooting, more or less paroxysmal and limited to the course of a nerve, and may be excited or aggravated by peripheral irritation. Tender points (points of Valleix) will be found where the sensory nerves make their exit from the bony foramina. The pain of headache is not confined to the distribution of any particular nerve and is constant. Symptomatic headache must also be distinguished from MIGRAINE (See p. 772). After eliminating these two conditions a careful search for one of the causes mentioned must be made.

Neuralgia

Neuralgia is a term applied to pain in the course of a nerve not caused by organic lesion.

Neuralgias have been divided into *idiopathic*, or those in which no cause can be found, and *symptomatic.* The latter are more frequent. Any of the sensory nerves may be the seat, but the fifth, sciatic, and intercostal nerves suffer most frequently. The cervico-occipital, lumbo-abdominal, crural nerves, and those of the brachial plexus are also often involved.

Pathology.—With greater knowledge cases of functional neuralgia are becoming more rare. Neuralgic pain, however, may be caused by many organic causes and these, for purposes of differential diagnosis, are here included. Most cases are due to a mild perineuritis; those occurring after middle life to arteriosclerosis and insufficient blood supplied to the nerve trunks. In some cases of tic douloureux, degeneration of the nerve fibers and Gasserian ganglion cells have been found associated with arterial disease. In toxic cases the nerves may be irritated without organic change occurring but in many of these there is probably some neuritis. It must not be forgotten that pain of the neuralgic type is due to irritation of the posterior nerve roots either by meningitis, either spinal or in the cerebello-pontile angle, vertebral disease or the lesions of tabes dorsalis. Disease of the Gasserian ganglion will also cause it.

Etiology.—Those possessing a gouty diathesis are especially liable. It is more common in cold or damp climates and in winter than in summer.

The possible **causes** of neuralgic pain to be considered are:

1. *Toxemic.*

(*a*) Metabolic, as uremia, gout, rheumatism, constipation, indigestion.

(*b*) Infectious, as influenza, malaria, and other infectious diseases.

(*c*) Metallic; lead and other metallic poisons.

2. *Vascular;* as anemia, arteriosclerosis, fatty heart.

3. *Reflex;* eye strain, carious teeth, non-erupted teeth, disease of accessory nasal sinuses.

4. *Neuroses;* neurasthenia, in fact debility from any cause; hysteria.

5. *Organic;* tumor of Gasserian ganglion; tumor or meningitis in cerebellopontile angle, disease of vertebra, meningitis, spinal tumor, tabes dorsalis.

Symptoms.—As has been said, the pain is sharp, burning, shooting and paroxysmal in type. Between the paroxyms there may be a constant dull pain. It is increased or excited by irritation, especially by movement of the parts. There is often hyperesthesia in the region of the affected nerves, but continued firm pressure may give relief. In some cases tender points may be found which correspond to the exit of the nerve from a bone canal or muscle or fascia (points of Valleix). Vasomotor, secretory, and trophic disturbances may occur, especially herpetic eruptions; in neurotic individuals painful impressions sometimes remain after the actual condition has been cured—this is termed *"reminiscent or hallucinatory neuralgia."*

Differentiation.—Neuralgia must be distinguished from HEADACHE and NEURITIS. The differential points between it and headache have been mentioned on page 573. As has been said, many cases called neuralgia are really a mild neuritis. It differs from typical neuritis as follows: In this the pain is constant without shooting exacerbations; there is *tenderness over the course of the affected nerve* made worse by pressure and there may be anesthesia, motor paralysis, muscular atrophy, and loss of or diminished reflexes in the parts supplied by the affected nerves.

Tic douloureux.—One of the most frequent seats of neuralgia is the fifth nerve. It has been termed, "tic douloureux." This may be due to any of the causes mentioned on page 573, but is most frequently found in people past middle life and in whom arteriosclerosis is present. The pain is of the character described above, and during the paroxysms twitching of the facial muscles frequently occurs with profuse lacrimation and secretion from the nasal mucous membrane, sweating and dilatation of the vessels of the conjunctiva. All the branches of the nerve may be affected or the condition may be limited to one or two of them.

Involvement of the *ophthalmic division* causes pain in the supra-orbital region (sometimes termed brow ague, owing to its frequently being caused by malaria), eyelid, eyeball and inside of nose. Tenderness is found at the supra-orbital notch.

The *superior maxillary division* causes pain in the face between the orbit and the mouth, the side of the nose and upper teeth. Tender points are found at the infra-orbital foramen, along the upper gum and over the prominent part of the malar bone.

When the *inferior maxillary division* is affected the pain is felt in front of the ear, the chin, just above the parietal eminence, the lower teeth and the side of the tongue. Movements of the jaw or tongue are liable to aggravate the paroxysms. Herpetic eruptions may occur over the course of the nerves. It is important to determine which branch or branches are involved for therapeutic reasons. When due to either tumor in the cerebellopontile angle, or disease of the Gasserian ganglion, anesthesia will usually be found in addition to the pain (Fig. 150). It is important to remember that bulbar tabes may cause pain of this character, in which event other symptoms of tabes will be found.

Intercostal Neuralgia or Pleurodynia.—This condition has been described on page 529. It frequently precedes the development of herpes zoster or shingles, or may be due to irritation of the posterior roots from tumor or meningitis or tabes.

Sciatica is in the vast majority of cases a neuritis and will be described on page 665.

Cervico-occipital Neuralgia.—Cervico-occipital neuralgia means pain in the distribution of the first four cervical nerves, especially the great occipital branch. In addition to the general causes mentioned (p. 573) it may be due to cervical caries if high up. The pain is felt in the occipital and sometimes in the posterior parietal region. It may be associated with neuralgia of the fifth pair. The scalp may be tender.

Cervicobrachial and Brachial Neuralgia.—Cervicobrachial and brachial neuralgia or pain confined to the distribution of the four lower cervical and first dorsal nerves may be caused by any of the various causes given on page 573; even decayed teeth have been accused. Most cases are organic in origin. Oppenheim gives the following as causes of such pain: commencing tumor in the cortical arm centers, commencing sarcoma of the humerus, high tabes, cervical hypertrophic pachymeningitis, vascular or vasomotor spasm associated with arteriosclerosis, angina pectoris, referred pains from disease of viscera, gallstones, vertebral caries, occupation neurosis and neuritis, to which may be added cervical rib, and tumor of the cervical region of the cord.

Lumbo-abdominal Neuralgia.—Lumbo-abdominal neuralgia, or pain in the distribution of the first three lumbar roots and one-half the fourth, may be due to tabes, lumbar pachymeningitis, tumor in the lower dorsal region of the cord, disease of pelvic viscera, vertebral caries, arteriosclerosis, neuritis or injury of the nerves and myositis of the abdominal muscles. In the latter case there will be *diffuse* tenderness over the muscles and aggravation of pain when they are made to contract.

Meralgia Paresthetica.—Meralgia paresthetica, or neuralgia of the external cutaneous nerve of the leg, is a condition of pain and paresthesia in the outer aspect of the thigh, usually induced by standing or walking. A case has been reported due to the pressure of a corset. The region supplied by the middle cutaneous nerve may also suffer.

(*c*) *Paresthesia*

Paresthesia, or *dysthesia,* is the term applied to perverted sensations, such as tingling, crawling, itching or pruritus, numbness, etc.

They may be due to organic disease anywhere in the sensory tract and in many cases seem to be of functional origin. Toxemia is a frequent cause and in the case of itching especially may be due to cutaneous irritation. Cases of tabes dorsalis, among organic diseases, are especially liable to suffer from it. Either degeneration or spasm of the arteries may also be a cause.

Acroparesthesia

A characteristic form is known as acroparesthesia, or *waking numbness.* This occurs most frequently in women about middle life, who have their hands in water a great deal or who sew considerably.

Symptoms.—The symptoms consist of numbness and tingling in the hands and sometimes in the feet. This is especially apt to be severe after waking in the morning and it sometimes may be so severe as to awaken the patient. The fingers are apt to feel clumsy, and some cyanosis may be present.

Differentiation.—In making the diagnosis all of the causes above mentioned must be considered. From NEURITIS it is distinguished by the absence of tenderness over the nerve trunks and of motor and sensory paralysis and muscular atrophy; from RAYNAUD'S DISEASE by the absence of pallor of the afflicted parts followed by congestion.[1] It must be remembered that the first symptom of the degeneration of the cord which may be caused by PERNICIOUS ANEMIA and other toxemiae and dyscrasiae is paresthesia of the hands and feet. The patient should also be examined for symptoms of TABES DORSALIS, NEURASTHENIA, and HYSTERIA.

4. Symptoms Due to Destructive Lesions of the Sensory Tracts

These differ according to the pathway diseased, i. e., either *touch, pain, temperature, muscle sense* (p. 556), or *special senses* (p. 597). They may be all damaged, or one or more may be and the others not.

Sensory fibers in the peripheral nerves and spinal cord seem to be less vulnerable to injury than the motor fibers. Lesions equally affecting both motor and sensory tracts will often destroy motor functions and affect the sensory but little or not at all, and when these are damaged they recover their function sooner than do the motor.

Destructive lesions, if severe enough, cause loss or diminution of the conducting power of the tracts affected. Such lesions may be either organic or functional.

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[1] Acroparesthesia is thought by many to be an abortive form of Raynaud's disease.

Loss of Tactile Sensibility.—Loss of tactile sensibility is called *anesthesia.* When the loss involves one-half the body it is termed hemianesthesia.

The face is not always affected. When complete, the entire lateral half of the body, including mucous membranes and special senses, is involved. Such a condition is practically only found in hysteria or in an organic lesion with hysteria coexisting. Anesthesia, with loss of all the special senses, does not occur from an organic lesion, nor is the line of demarcation between normal and lost sensibility so sharply limited to the middle line of the body (pp. 620, 622, 784).

Crossed hemianesthesia means anesthesia of one side of the body and of the opposite side of the face. By *hypesthesia* we mean diminution but not complete loss of the power of perceiving sensations of touch.

Loss of Sensibility to Pain.—Loss of sensibility to pain is known as *analgesia.* If limited to one lateral half of the body it is termed *hemianalgesia.* Analgesia may exist alone or be associated with other forms of sensory paralysis. In some diseases (notably tabes dorsalis), instead of loss there is delay in the perception of the stimulus, so that it is not felt until an appreciable interval (as much as several seconds) has elapsed after its reception. This is sometimes termed *"delayed sensation."*

There may also be loss of power of correctly locating the point stimulated. This may vary from an inch or more to a corresponding point on the other side of the body (*allochiria,* et seq., p. 567).

Loss of Temperature Sense.—Loss or impairment of temperature sense is usually associated with that of pain (p. 556). It may consist of either inability to tell hot from cold or hot objects may give the sensation of cold, cold being recognized, or vice versa. Sometimes marked differences in temperature can be recognized and slight ones cannot (p. 578).

Dissociation of Sensation.—Owing to the location of the tracts conducting sensations of touch and those of pain and temperature (p. 556) dissociation of sensation is a not uncommon symptom. Usually tactile sensibility is preserved and sensibility for pain and temperature is lost. This is most commonly found in syringomyelia, but may occur in other lesions, either about the central canal of the cord or affecting the posterior nerve roots, viz.: hemorrhage into the cord (hematomyelia), spinal tumor, multiple sclerosis, myelitis, vertebral caries and inflammation, pachymeningitis, tabes, diseases of the peripheral nerves and hysteria.

Muscle Sense.—What is generally known as muscle sense includes a number of different forms of sensibility due to impressions received from muscles, tendons, and joints, viz.: recognition of the direction of active and passive movements, of the position of the limbs and of pressure and resistance.

Vibrating Sensation.—Another form of sensation, the presence or absence of which may be useful in diagnosis, is the so-called *vibrating* sensation. By this is meant the feeling of vibration or trembling that is felt when a vibrating tuning fork is placed over subcutaneous bony prominences in any part of the body This sensation is probably conducted from the periphery by the posterior columns and therefore is lost in diseases affecting either these columns, the posterior nerve roots or the peripheral nerves. It may be lost very early.

STEREOGNOSTIC SENSE.—The stereognostic sense is the name given to memories preserved in the cortex of the parietal lobe (Fig. 135) of the characteristics of objects by which we are enabled to recognize them by their shape, feel, weight, etc. It therefore depends upon tactile impressions and those of muscle sense, the latter being probably the more important. Impairment of this power is termed *astereognosis* and is a form of *agnosia* (p. 617).

By some the term astereognosis has been limited to loss of the power of recognizing the shape, size, and form of an object in three dimensions, while loss of the power of telling what the object is is termed *asymbolia;* the former may be preserved while the latter is lost.

Methods of Examination.—In making these tests, if the lesion is in the peripheral nerves, it is important to remember the results of the researches of Head, Rivers and Sherren (Brain, 1905), who showed that these nerves contain three systems of afferent fibers:

1. Those which subserve deep sensibility and conduct the impulses produced by *pressure* and *movement of limbs.* The fibers of this system run mainly with the *motor nerves* and are *not* destroyed by division of the sensory nerves of the skin. These fibers also are located in the tendons. If the pressure is severe enough pain may be caused.
2. Those which subserve *protopathic sensibility,* i. e., painful stimuli and extremes of heat and cold, but inability to localize the spot stimulated.
3. Those which subserve *epicritic sensibility,* i. e., the power of cutaneous localization, of the discrimination of two points, and of the finer degrees of temperature, as cool and warm. Protopathic sensibility returns first after a sensory nerve has been injured.

When the lesion is in the *spinal cord* the phenomena differ as follows:

1. If sensibility to pain is abolished all forms of painful stimuli are simultaneously affected.
2. Sensibility to heat may be abolished without coincident disturbance of that to cold, and vice versa. When sensibility to heat is disturbed in consequence of an intramedullary lesion the patient no longer appreciates any thermal stimulus between 30° and 60° C. That is to say, insensibility is absolute for both intermediate and extreme degrees. This may be absolute and yet the patient may be able to recognize the lightest tactile stimulation and to discriminate the two points of compasses.
3. If sensibility to touch is abolished in consequence of an intramedullary lesion, all forms of tactile stimuli will be affected. The peripheral afferent impulses for touch and pressure arriving by way of the epicritic and deep systems (*supra*) become combined in the spinal cord.

4. In intramedullary lesions every other form of sensibility may be abolished in a part which still remains sensitive to *passive movement.* Or the patient may be unable to recognize even the greatest passive movements, although sensitive to all tactile and painful stimuli; or the sense of passive movement and position may be disturbed in one leg, while every other loss of sensation may be formed in the leg of the opposite side.

5. Within the spinal cord the impulses which underlie the power of discriminating two points are separated from those of tactile sensibility (Head & Thompson, Brain, 1906).

It is also important to remember that *areas of lost sensibility* differ according to whether the lesion involves either the posterior nerve roots coming from one or more segments of the cord (segmental type, p. 627), the peripheral nerves (peripheral type, pp. 656, 657, 659, 660), the conducting apparatus in the cord (p. 627), or is central or psychic, which occurs in hysteria (p. 777, Fig. 123).

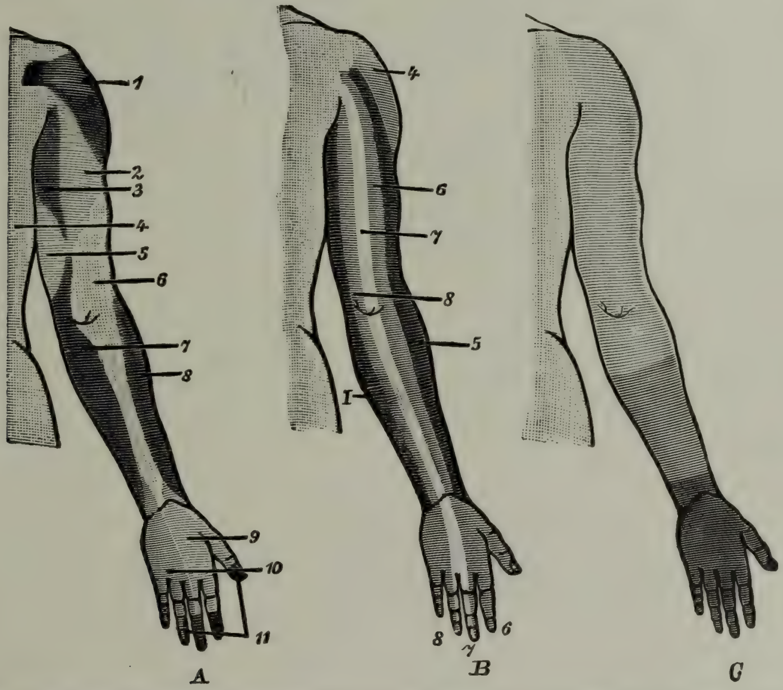

Fig. 123.—The Three Types of Disturbances in Sensibility at the Extensor Side of the Right Upper Extremity. (After Obersteiner and Redlich.) A, Peripheral Type; 1, N. supraclavicularis; 2, N. axillaris; 3, N. Cutan. Post. Su. radialis; 4, Lateral Branches N. Spinal; 5, N. Cutan. Medial radialis; 6, N. Cutan. Post. Inf. radialis; 7, N. Cutan. Med.; 8, N. Cutan. lateralis; 9, N. radialis; 10, N. ulnaris; 11, N. medianus. (After Bernhardt). B. Segmental Type, 4, 5, 6, 7, 8, 4-8, Cervical Segment; I, 1. Dorsal Segment. (After Allen Starr.) C, Central Type. (After Curschmann's "Textbook of Nervous Diseases," published by P. Blakiston's Son & Co., Philadelphia.)

Before testing for sensory loss the patient should have his eyes covered. *Loss of tactile sense* is tested for by touching the part with a very light object, as a piece of cotton wool or camel's hair brush. As little pressure as possible must be made (p. 578). Whatever is used is drawn over the body and limbs, beginning at the top and going down, or vice versa.

The patient is asked to tell when he feels anything and when he does not, the latter spot being marked; the procedure is then repeated, coming in the opposite direction and also from the sides. In this way the area is mapped out. It is well to take the testing object off the patient at intervals to see if he is paying attention and recognizes when he does not feel it.

The *power of localization* must also be tested (p. 567). For delicate observations various forms of instruments, as the v. Frey hair esthesiometer, may be used.

Loss of pain sense is discovered by sticking the parts with a sharp object, as a needle or pin. The body is gone over as in testing for anesthesia. It is important to remember that in some diseases the stick of the pin may be recognized as touch and pressure. Therefore the patient must indicate that it is the painful sensation he feels.

A good plan is to instruct the patient to count out loud each prick of the pin that he perceives; if he misses several counts we know that he does not feel it.

It must also be noticed if there is delay in perception (p. 577). This may be determined by making the stimulus and at once removing the object, telling the patient to indicate when he feels it.

It must also be noticed if he is able to localize the place stimulated and distances apart at which two points can be recognized (pp. 567-578).

Instruments have also been devised for the delicate testing and comparison of pain sense.

Loss of temperature sense is discovered by using test tubes filled with hot and cold water of different extremes of temperature. Ice may be used for the testing of cold (p. 578).

Various functions must be tested in estimating the *loss of muscle sense;* only some or all may be interfered with.

We test the *perception of passive moments* by changing the position of part or all of a limb and instructing the patient to tell you when he feels the movement and the direction in which it is moved. It is best to first test the fingers and toes. In some cases the movement may be recognized but not the direction of it.

The *sense of position* is tested by placing a limb or part of it in a certain position and instructing the patient either to place the opposite one in the same position or to describe it. Another method is to ask him to touch with the normal hand some definite place on the suspected limb, as the knee, toe or finger.

Perception of pressure and resistance may be determined by using rubber balls of the same size filled with different quantities of shot. These are placed on the hand or leg and the patient told to indicate which is the heavier. A difference of about one-fortieth of the total weight should be detected if the perception is normal. Objects of different weight may be used to test the power of appreciating weight and differences of it. As has been said, great pressure may cause a painful sensation when pain sense for other stimuli is absent (p. 578).

Vibrating sense (p. 577) is tested by placing a large tuning fork over bony prominences, palms of the hands and soles of the feet. The patient must be asked what he feels; if he answers, "a vibrating or trembling sensation," we know that the sense is present.

The presence or absence of the *stereognostic sense* (p. 578) is detected by placing familiar objects in the hands and requesting the patient to name them. He should also be asked to describe the shape, consistency, and roughness or smoothness of the object.

Incoördination—Ataxia

Every movement of our body and limbs is due to the synchronous action of several muscles. Thus when we use the flexor muscles of a limb the extensors act as a balance (p. 564), otherwise the movement would be made in a jerky, irregular manner.

Synergy.—The performance of a direct movement, therefore, requires the delicate coöperation of the different muscles involved. This mechanism has been termed *synergy* or *synergia.* The more complex the movement the greater the demand upon this regulating function. A comprehensive definition is as follows:

> "Synergy is the power or faculty by which movements, more or less complex, but functionally definite, are associated in a special act or acts. It is motor association being carried out in movements of different parts of the same limb or in synchronous movements of the trunk and limbs," etc. (Mills and Weisenburg).

If for any reason the action of different muscles entering into a movement is not timed accurately, or if the function of one or more of them is lost, *asynergy* results and this causes incoördination.

The part of the brain which controls synergy is the cerebellum. Sensations travel to it through the posterior columns and direct cerebellar tracts from the muscles, articular surfaces, and tendons, and possibly to some extent from the skin. Interruption of these pathways causes incoördination. Usually, therefore, it is due to either spinal or cerebellar disease and rarely to cerebral disturbance.

Disease of the peripheral nerves sometimes causes incoördination, due to the interference with the transmission of the impulses from the muscles, etc. (*supra*), to the spinal cord. According to the **location of the lesion** the symptoms differ:

Cerebral.—Cortical lesions of the parietal lobe cause more or less incoördination of fine movements, as shown in trying to touch the end of the nose with the end of the index finger of each hand alternately, the eyes being closed (finger to nose test). In such cases, on the side opposite the lesion, a coarse, irregular tremor of the hand occurs.

Cerebellar.—Lesions here cause the most marked evidences of asynergy. These differ according to the part of the cerebellum involved, i. e., vermis or lateral lobes. Attempts have also been made to locate definite regions in the vermis and lateral lobes which control the movements of certain parts (*infra*).

Generally speaking, involvement of the vermis causes a disordered gait, known as *titubation.* This resembles somewhat that of alcoholic intoxication.

There is a tendency to fall in various directions. If a lateral lobe is involved, usually, but not always, the patient tends to fall toward the affected side.

The disordered gait is due to the fact that there is asynergy of the muscles which have to do with maintaining the erect posture, this causing the trunk to incline to either one side or forwards or backwards. The legs endeavor to follow the direction of the trunk in order to maintain the equilibrium; thus if the trunk leans forward and to the left the swing of the right leg will be much greater than that of the left, and vice versa. There may also be swaying of the body while standing or sitting (static ataxia); this is not, however, increased by closing the eyes (p. 587).

Mills and Weisenburg believe that the upper part of the vermis controls the shoulder, girdle, and upper part of the trunk and the lower part controls the pelvic girdle and lower part of the trunk. The difficulty in walking is greater when the lesion is in the latter place. If the patient is placed on the floor, on his hands and knees, the shoulders will sway and deviate to the side of the lesion if it is in the upper part of the vermis, while the pelvis will do this and the shoulders will remain quiet if the lesion is in the lower part. The pointing test of Bárány (p. 584) is useful to determine if cerebellar function is present or not.

When one lateral lobe is the seat of the lesion, other evidences of incoordination are manifest on the side of the lesion. Thus if the patient moves the limbs of this side certain phenomena occur, known as *dysmetria,* shown in the arms by the fact that when the finger to nose test is attempted the finger instead of touching the end of the nose shoots past it (sometimes termed either *hypermetria or hypermetry*).

In this it differs from the movement described above. Also the movement is made from the shoulder in a much wider arc and either quicker or slower than the normal person makes the movement. This can be shown in the legs by placing the patient in a recumbent position and asking him to put his heel on the opposite knee or some other point.

On the affected side there will be overflexion of the thigh on the body and overshooting of the desired point.

Another test is to require the patient to draw the heel up to the buttock and then straighten the leg on the abnormal side. It will be noticed that this is not done smoothly; the movement is quicker; the thighs more flexed on the abdomen and as the heel nears the buttock a to-and-fro sway of the limb occurs. Normally the flexion and drawing up of the leg will be done in one movement. If asynergy exists in addition to the phenomena mentioned, the hip will be flexed first and the extended leg raised abnormally high; with a second movement the leg is flexed, and in a third movement it is drawn up. Extension or straightening the leg will be done in the inverse order.

Another symptom is that known as either *adiadochokinesis* or *adiadochokinesia.* This is shown by the inability of the patient to alternately pronate and supinate the forearm quickly and regularly and in the lower limbs by inability to similarly alternately abduct and adduct the limb.

Incoördination of the *muscles of articulation* causing a jerky form of speech may occur. In fact, all coördinate muscular movements may be affected. Similar symptoms are caused by lesions of the cerebellar peduncles (p. 721).

NYSTAGMUS.—Mills and Weisenburg believe that nystagmus (*infra*) is a manifestation of asynergy of ocular movements, the center for which they place in the upper part of the vermis.[1]

By nystagmus is meant the rhythmic oscillation of the eyeballs which may be either lateral, rotary or up and down. When either the labyrinth or nystagmus circuit is at fault (*infra*) the movements are slow in one direction and quick in the opposite. When we speak of nystagmus being in a certain direction, we mean in that of the quick movement. In labyrinthine disease this is toward the diseased side. In cerebral disease the direction is indefinite.

Nystagmus is an important symptom in diseases of the cerebellum and vicinity, and its presence, absence or peculiarities in its character when present are now used in the localization of such lesions. For this purpose the tests devised by Bárány and Neumann are used. In the space at command a complete description of these cannot be given, but a résumé is essential. For a fuller description the reader is referred to recent works on otology.

That part of the nervous system concerned in these phenomena begins in the semicircular canals and from hence by means of the vestibular nerve (p. 645) to the nuclei of Deiters, von Bechterew, and the triangular nucleus in the medulla. Here the tract divides, one division going upward by means of the posterior longitudinal fasciculus through the pons to the nuclei of the third, fourth, and sixth cranial nerves. This may be called the *nystagmus circuit.* The fibers of the other division, or cerebellar circuit, run from the medullary nuclei through the restiform body to the cerebellar nuclei (emboliformis, globosus, and fastigii) of the same side and from them to the same nuclei on the opposite side, then through the superior cerebellar peduncles to the crus cerebri and cerebral cortex. Here an efferent tract begins which runs back to the cerebellum through the red nucleus, tegmentum and superior cerebellar peduncles, and from the cerebellum to the pyramidal tracts. According to Randall and Jones (Amer. J. Med. Sci., April, 1916), these fibers come from the horizontal canals, while those from the vertical canals go to the posterior longitudinal fasciculus and enter the cerebellum by the middle peduncle.

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[1] Grey, of Boston, has recently shown that cerebellar lesions may exist without nystagmus being present.

Nystagmus may be spontaneous or involuntary or induced by movements of the eyeballs in various directions or by stimulation of the nystagmus circuit. The former may indicate disease of the cerebellum itself, the latter extracerebellar disease.

To stimulate the nystagmus circuit either the turning or caloric tests are used. In the former, if the patient is turned slowly in a revolving chair a number of times to the right with the head at an angle of 30° forward, horizontal nystagmus to the left is produced. This consists of a slow movement in the direction of the turning and a quick movement (cerebral recoil) in the opposite. If the patient is turned to the left the nystagmus is in the opposite direction.

Similarly, if the right ear is douched with cold water (68° F.), with the head inclined 30° forward, rotary nystagmus to the left is caused. If the head is held 90° backward, horizontal nystagmus to the left is produced. Douching the left ear in a similar way causes nystagmus to the right.

Similar movements of the eyeball can be caused with the constant current (p. 610).

A destructive lesion anywhere in the nystagmus circuit will either prevent or diminish these reactions according to the severity. An irritative lesion causes an increased movement. This may result from lesions outside the circuit, as the cerebellum, making pressure upon it. If there is destruction of the auditory nerve, a spontaneous slow movement to the side of the lesion will occur, not increased by stimulation. Deafness of nerve origin will also be present.

In labyrinthine inflammation, if mild, the slow movement is to the same side and is not increased by stimulation; if the disturbance is severe the opposite is the case. Some deafness may be present. In lesions elsewhere deafness is absent.

Destructive lesions of the cerebellar circuit cause either absence or perversion of *"past pointing."* This depends on the fact that normally one can locate with the eyes closed a point in space that he has previously touched or seen the location of. If, however, the patient is either turned or douched, as above described, he is unable to do this for the reason that vertigo is also caused by these proceedings and he loses his relation to the object (p. 646).

The method used is to place the patient in front of you with his eyes closed; have him place the index finger of the right hand on your finger; then ask him to elevate the arm (held stiffly) and bring it down upon your finger again. Normally this can always be done. If, however, the patient is turned to the right he will "past point" to the right; that is, his finger will go to the right of the examiner's finger. Similarly, if turned to the left he will "past point" to the left.

Again, if the right ear is douched with cold water (68° F.), with head inclined 30° forward, he will "past point" to the right. If the left ear is douched he will "past point" to the left. Hot water (112° F.) causes the opposite of these reactions.

If, however, there is interference with the cerebellar circuit, he will either locate the point correctly or if he does "past point" he will do so, either to a limited degree or point in the wrong direction. If the lesion is in the afferent part of the circuit, vertigo will not be caused; if in the efferent part, vertigo is caused.

In lesions, therefore, of the cerebellum or its superior or inferior peduncles, absence of "past pointing" may be a valuable diagnostic sign in distinguishing them from pons lesions, in which there may be some incoordination from involvement of the middle cerebellar peduncles or pressure on the cerebellum. In the former nystagmus will be present. In the latter nystagmus will be absent or lessened and "past pointing" will be normal. Deafness would not be present as it would in lesions affecting the eighth nerve or labyrinth.

Nystagmus, in addition to being a symptom of cerebellar disease, is also a symptom of multiple sclerosis, Friedreich's ataxia, irritative conditions of the internal ear, and destruction of the eighth nerve. In lesions of the internal ear it is spontaneous or involuntary. It also occurs in miners, those with high myopia, and may be congenital.

In the organic conditions mentioned it is of the vestibular type (*supra*). When due to ocular conditions alone the rapidity of motion is the same in both directions.

In the following table an attempt is made to give the phenomena observed in lesions of the different regions in which the above tests may be of use. It is not absolute, as pressure on different regions may modify them. In doubtful cases the examination should be made by one familiar with the tests in their application to neurology. The table on page 723 should also be consulted for further differential symptoms.

While tumors in the cerebellopontile angle do not always arise from the auditory nerve, they most frequently do. The phenomena mentioned would not be present in those which do not (See p. 722).

	Cerebellum	Cerebellopontile Angle, Auditory Nerve	Pons	Labyrinth
Nystagmus before stimulation of vestibular nerve	Spontaneous nystagmus may or may not be present. It usually is	May or may not be present. Usually spontaneous if present	May or may not be present. Often only present when eyeballs are moved	In acute cases of inflammation spontaneous nystagmus present, which gradually diminishes in severity. In chronic cases usually absent
Nystagmus after douching or turning	Increased	Not increased	May be absent or weak	Not increased
Past pointing	Absent or points to wrong side	Absent	Is present if the horizontal canal is stimulated by turning with head at 30° forward, or by cold douching with head 90° backward Is absent if the vertical canals are stimulated by turning with head 90° backward or douching with head 30° forward	Is absent or to wrong side, or does not point as far past the point as he should
Hearing	Good	Diminished or absent	Good	Diminished or absent
Vertigo	Not marked. Subjective rotation of self from side of lesion	Paroxysmal attacks; subjective rotation of self to the side of tumor. Tinnitus aurium	Usually absent; may be slight.	Paroxysmal attacks. Tinnitus aurium.
Symptoms of asynergy	Present and well marked	Usually present, but not so marked as in intracerebellar tumors	May be slight or absent	Absent

Spinal and Peripheral Nerves.—Incoördination from lesions here is due to interference with the impulses mentioned on page 581, reaching the cerebellum and cerebrum, and is that usually known as *ataxia.* In the case of the former, the lesion is principally in the posterior columns although degeneration there may primarily have been in the posterior nerve roots (See Tabes dorsalis). The disorder is usually manifested most in the gait, the patient walking with the legs wide apart, the foot raised higher than normal and thrown forward in a jerky manner and the heel brought to the ground with a sudden stamp. The eyes are fixed on the ground (Fig. 124).

This is made worse or sometimes may be manifest only when either the eyes are closed or the patient is in the dark. In some cases more difficult movements, as walking backwards, or standing on one leg, may be necessary to bring out the condition. This is due to the fact that, for the reason mentioned above, the patient does not know the position of his limbs; this is compensated for by the use of the eyes; therefore if deprived of the use of these, the symptoms become worse.

Static ataxia is here manifested with the eyes closed. It is brought out by causing the patient to stand with the feet close together and the eyes closed. Marked swaying will usually occur and he may fall. In milder cases the symptom is indicated by the marked muscular effort the patient requires to maintain the equilibrium. This is known as *Romberg's symptom.* If the arms are affected there is difficulty in performing delicate movements with the fingers, as buttoning the clothes. The finger to nose test is performed very much as in cerebral disturbance (p. 582). The heel to knee test (p. 582) is also not performed normally. Ataxia confined to the finer movements of the hands and fingers (acroataxia) is characteristic of that due to disease of the peripheral nerves (multiple neuritis) and also of that due to degeneration of the cord found often in pernicious anemia (p. 702). In spinal ataxia muscles nearer the trunk are usually first affected (proximo-ataxia).

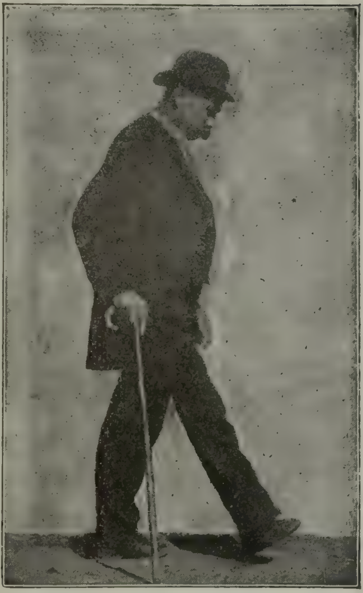


Fig. 124.—Gait in Tabes. Observe Overextension of Supporting Knee, Rigidity of Advancing Leg, Elevated Toe, Heavily Descending Heel, Watchfulness of Steps, and Assistance by Cane. (After Church and Peterson's "Nervous and Mental Diseases," published by W. B. Saunders Co., Philadelphia.)

5. Reflexes

By a reflex we mean an involuntary muscular contraction due to a peripheral stimulation of a sensory nerve. It depends upon a so-called reflex arc, consisting of a sensory or afferent nerve fiber, the reflex center in the anterior horn of the spinal cord or cranial nerve nuclei, and an efferent or motor nerve fiber. The location of the different reflex arcs is given in the table (p. 625).

The activity of the reflexes, especially the tendon (*infra*), is maintained by muscle tone. This depends in large measure upon sensory impressions transmitted to the cerebrum through the sensory nerves and sensory tracts and from the cerebrum to the muscles by the extrapyramidal tracts. Muscle tone and the deep reflexes are controlled by cerebral influence (p. 558).

Reflexes are divided into *skin or superficial, tendon and muscle or deep, and visceral.* If any part of the reflex arc is destroyed the corresponding reflex is lost, if the extrapyramidal tract (p. 556) is destroyed the tendon reflexes are lost and muscle hypotonicity also occurs. If cerebral inhibition is interfered with, as in lesions of the upper motor neuron, the tendon reflexes are increased and muscle hypertonicity is present (p. 558). Sometimes irritation of a peripheral nerve, as in the early stage of a neuritis, will cause increase in the corresponding tendon reflex; but it soon becomes diminished or absent.

(a) Skin Reflexes

These are produced by scratching, tickling, or irritating the skin, which if the reflex is present causes a contraction of the muscles near the irritated part. If the limbs are irritated there is an involuntary effort to remove the part from the irritation by contraction of the flexors (defense reaction). The skin reflexes are many and not all of them are always present in normal people. Their presence or absence may be of service in localizing lesions in the spinal cord (p. 625).

The most important are:

Plantar Reflex.—Normally when the sole of the foot is irritated, the leg is drawn away by flexion of the thigh on the body and the leg on the thigh; in addition the foot and toes are plantar flexed. This is known as the plantar reflex. It is not always so fully developed as this and may consist merely of plantar flexion of the toes. When, however, there is a *lesion of the fibers in the pyramidal tract* that supply the leg, the toes instead of being plantar flexed are dorsal flexed or extended; this is especially so of the great toe.

To be typical it should be extended slowly with some separation of the other toes. This is known as the *Babinski reflex or Babinski toe sign.* In some cases in which the lesion is slight the toes may be immovable; this is also pathological. The extensor response is normal in children under two years of age. To elicit the sign the patient should be recumbent, the leg in a position of relaxation, and the foot warm; a moderately sharp instrument, as a tooth pick, should then be stroked from the heel to the toes along the inner side of the foot.

Sometimes the reflex may occur spontaneously; this is due to a similar cause. A similar action of the toes in a similar pathological condition is seen in *Oppenheim's and Gordon's reflexes.* The first is elicited by drawing a blunt instrument down the lower third of the inside of the leg just back of the tibia, the second by grasping the relaxed muscles of the calf and squeezing them.

Cremaster Reflex.—The cremaster reflex is caused by irritating the skin in the inside of the thigh, when a drawing up of the testicle will be seen.

Abdominal Reflex.—The abdominal reflex is shown by contraction of the rectus muscle of the abdomen when the side of the abdomen is scratched. The upper quadrants may react when the lower will not, and vice versa (p. 628).

Epigastric Reflex.—The epigastric reflex is shown by contraction of the upper fibers of the rectus muscle when the skin of the lower part of the thorax is irritated or the costal cartilages on either side of the xiphoid depression are tapped.

Lid or Conjunctival Reflex.—The lid or conjunctival reflex is shown by closure of the eye when either the conjunctiva, cornea, or retina is irritated.

Pupillary Skin Reflex.—The pupillary skin reflex is caused by pinching the skin on the side of the neck when dilatation of the pupil takes place.

Pharyngeal Reflex.—The pharyngeal reflex is shown by gagging and elevation of the soft palate when the back of the pharynx and faucial pillars are irritated.

Supra-orbital Reflex of McCarthy.—The supra-orbital reflex of McCarthy is caused by striking the supra-orbital foramen with the end of the finger or a percussion hammer when a contraction of the orbicularis palpebrarum will be seen. Its absence indicates destructive lesion of either the supra-orbital or facial nerves.

Deep or Muscle and Tendon Reflexes

Only the most important are mentioned. It must be remembered that their absence may be caused by mechanical reasons, such as either ankylosis of joints or excessive spasticity of the muscles, they being so contracted that further contraction is not possible.

Knee Jerk or Patellar Tendon Reflex.—The knee jerk or patellar tendon reflex is practically always found in normal individuals; very rarely it is absent in such. Either its increase, diminution, or absence are indications of disease. It consists of a sudden contraction of the quadriceps femoris, vastus internus and externus muscles when the patellar tendon is struck while the leg hangs loosely at right angles with the thigh.

One leg may be crossed over the other, the patient may sit on the edge of a table, or if he is unable to sit up the leg may be raised up and supported at the bend of the knee. Complete relaxation must be had; it may be secured by making the patient look at some object at the time the knee is to be struck. The end of the fingers or a percussion hammer may be used to strike the tendon. The knee jerk is increased normally by any muscular contraction or excitement. This is utilized to endeavor to bring it into activity when apparently absent by causing the patient to perform some muscular movement, such as tightly closing the hands and pulling in his clasped fingers at the instant the tendon is struck. This is termed *reinforcement by Jendrassik's method.* It can rarely be done if the absence is due to organic disease, but if diminution is due to functional cause it may be increased. When there is great increase *patellar clonus* may be elicited; this consists of rapid clonic contractions of the quadriceps muscle when the patella is either suddenly pushed downward or being held in this position the finger holding it is percussed.

Front Tap Contraction.—In conditions of great reflex and muscle irritability, if the knee be extended and the foot dorsal flexed, a tap on the upper and outer portion of the leg over the common extensor and peroneal muscles causes contraction of the calf muscles.

Tendo Achillis or Ankle Jerk.—The tendo Achillis or ankle jerk is brought out by striking the tendo Achillis, the foot being slightly dorsal flexed, with the ends of the fingers or a percussion hammer. Contraction of the soleus muscle and plantar flexion of the foot occurs. If the patient is able to stand it is best elicited by placing him on his knees on a chair or bench, so that the feet just project over the edge, and then striking the tendon.

Ankle Clonus or Foot Clonus.—When the ankle jerk is very active ankle clonus or foot clonus will occur if the slightly flexed leg is grasped under the knee joint with one hand and dorsal flexion of the foot is suddenly made with the other.

A series of clonic contractions of the soleus muscle will occur, causing a rapid to-and-fro movement of the foot, which is maintained so long as the pressure on the foot is kept up. It can also be elicited by causing the patient, while sitting down, to bear the weight of the limb on the point of the foot.

True ankle clonus was supposed to only be present when there is degeneration of the pyramidal tracts. Usually this is so, but it has been found in certain toxic states and the author has observed it in a healthy man. (Am. J. Med. Sci., July, 1913, p. 1; also J. Nervous and Mental Diseases, Oct., 1912.) In functional nervous disease, as hysteria, ankle clonus may occur, but as a rule it consists under these conditions of a few oscillations and does not persist during the maintenance of pressure on the foot; such a clonus is called *pseudo-ankle clonus.*

Paradoxical Contraction.—Sometimes when there is a condition of muscle and reflex irritability, if the ends of a muscle are suddenly and passively approximated, tonic contraction of the muscle occurs; thus in such a case, if the foot is dorsal flexed, contraction of the tibialis anticus will occur.

Wrist Jerk.—The wrist jerk is elicited by striking the flexor tendons at the wrist, made prominent by passive extension of the hand, when flexion of the hand will occur.

Elbow or Triceps Jerk.—The elbow or triceps jerk consists of a contraction of the triceps muscle when its tendon is struck just above the elbow joint. The forearm should hang loosely at a right angle to the support.

Biceps Jerk.—The biceps jerk may be obtained by partially flexing the forearm on the arm, then placing the thumb of the supporting hand over the biceps tendon and pressing it downward. It is struck with the hammer or fingers of the other hand. Contraction of the biceps will result.

Contraction of Supinator Longus—Flexion of Elbow Joint.—Percussion over the end of the radius will cause contraction of the supinator longus and flexion of the elbow joint.

Jaw Jerk.—The jaw jerk is obtained by causing the patient to sit with the mouth partly opened. A ruler or something similar is laid on the teeth and struck. Contraction of the masseters results. This is never found in health.

Numbers of other reflexes may be excited by tapping different muscles. They are not of great importance in diagnosis.

The Visceral Reflexes

(b) Reflexes of the Eye

Direct Light Reflex.—One of the most important is the *light reflex*, produced by throwing a bright light into the eye. The iris should then contract and dilate when the light is removed. A small electric lamp is convenient. The light should be brought into the field from the side and close to the eye to eliminate the influence of accommodation; the other eye should be covered.

Consensual or Indirect Light Reflex.—If the patient is placed facing a window and one eye—the one which is being tested—is shaded, but so that the movements of the iris can be observed, and we then alternately cover and uncover the eye, the iris of the screened eye should contract and dilate with the exposed one.

Hippus.—Sometimes, especially in neurasthenics and some cases of cerebral syphilis, the pupil after contracting will dilate again, doing this alternately several times. This is known as hippus (a mild degree may be normal).

Rebounding Pupil.—In similar conditions a pupil may contract when exposed to light, then while exposed to the same light dilate and remain dilated. This is termed the rebounding pupil.

Wernicke's Hemianopic Pupillary Reflex or Inaction Sign.—Lesion of the tract (p. 597) posterior to the reflex centers does not cause loss of the light reflex (Fig. 127). If the lesion is in the tract of one side anterior to or involving these centers there is loss of the pupillary reflex when the light is thrown in on the blind half of the retina only.

Ciliary Reflex.—When the eye looks at a far object the iris dilates. When it looks at a near object it contracts. This is termed the ciliary reflex, or the convergence and accommodation reflex. Each eye should be tested separately, the other eye being covered as in testing the light reflex.

Argyll-Robertson Pupil.—Frequently in paresis and tabes dorsalis, and rarely in cerebral syphilis and multiple sclerosis, the light reflex is lost and the convergence reflex preserved. This is known as the Argyll-Robertson pupil. The phenomena may rarely be reversed.

In some cases the pupil may not react to either light or convergence (p. 635). This is found frequently in cerebral syphilis. An abnormal dilatation of the pupil is termed *mydriasis*. It may be present in neurasthenia as well as organic nervous diseases. An abnormally contracted pupil is known as *myosis or myotic pupil*. It is frequently found in tabes, syringomyelia, and conditions causing irritation of the cervical sympathetic (p. 594) and pontine hemorrhage.

In old people the convergence reflex may be sluggish normally. An irregular pupil is suggestive of syphilis (former syphilitic iritis).

(c) Reflexes of the Bladder, Rectum and Sexual Apparatus

Urination and defecation are reflex acts under the control of higher inhibitory centers in the cerebrum.

v. Müller and others believe that the reflex centers are not in the sacral region of the cord as is held by most authors (p. 624), but are in the pelvic sympathetic ganglia (hypogastric plexus). It is certain these have some influence.

The reflex arc consists of the nerves of the pudendal plexus, especially the pudendal nerve, and the third and fourth sacral segments of the cord. These nerves are closely connected with the sacral ganglion and hypogastric plexus of the sympathetic system (Fig. 126).

If the inhibitory function is lost there is no longer voluntary control of the acts of urination and defecation. As soon as the bladder is full it is emptied, although in the case of the rectum this is only so when the bowels are loose. If constipated or there is also loss of the expulsive power they are emptied involuntarily, but at long intervals. Disease of the pyramidal tracts causes interference with inhibition. If this condition persists, weakness of the detrusor muscles occurs after a time or they may have been weak from the beginning. Then the bladder is not completely emptied, residual urine accumulates, until finally the sphincter muscle is overcome and constant dribbling of urine occurs. This is known as the *incontinence of overflow,* or *retention.*

This may occur in retention from other causes, as stricture or enlarged prostate. If the centers in the sacral region of the cord (p. 628) are diseased, constant dribbling occurs without previous retention. When the sensory fibers are interfered with the patient does not recognize when the bladder or rectum is full or when evacuation is taking place; if only the motor functions are interfered with this is known. If the motor part of the reflex arc is diseased there is also constant dribbling as in lesions of the cauda equina (Fig. 125).

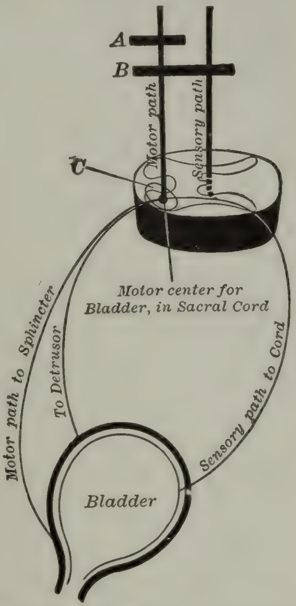


Fig. 125.—Diagram Illustrating the Innervation of the Bladder and the Effect of Lesions in Various Parts of the Spinal Cord upon the Function of Micturition. A Lesion, A, Which Interrupts the Voluntary Path to the Bladder Center in the Sacral Cord Causes Incontinence of Urine. When a Sufficient Quantity of Urine Accumulates in the Bladder There Occur a Reflex Contraction of the Detrusor and a Relaxation of the Sphincter. The Sensory Path from the Cord to the Brain Being Uninvolved the Patient is Conscious of the Process, But Cannot Exercise Voluntary Control Over It. With a Lesion, B, Which Involves Also the Sensory Path, the Patient Is Unconscious of the Filling and Reflex Emptying of the Bladder. A Lesion, C, Which Causes Destruction of the Sacral Reflex Center of the Bladder Causes Continuous Dribbling of Urine, and Not Its Automatic Expulsion at Intervals. (After Herter, from "Potts' Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

In the case of the rectum this condition can be distinguished from incontinence due to loss of inhibition by determining if the *anal reflex* is present or not. This is done by inserting a finger into the rectum. If present, the finger is grasped by the sphincter; if not, there is relaxation.

When there is merely weakness of the auxiliary muscles there is difficulty in starting the stream of urine or there may be obstinate constipation. The former may also be the case in urethral stricture or prostatic disease.

Damage to the reflex arc governing the *genital functions* causes loss of the power of erection of the penis and of the virile reflex (*infra*). Removal of the inhibitory influence may cause priapism. In normal men pinching the glans penis will cause a contraction of the perineal muscles which may be felt. This is known as the *virile reflex.*

6. Symptoms Due to Vasomotor, Secretory and Trophic Disturbances

The sympathetic system is closely related with the spinal cord and nerves. The cells of the intermediolateral tract are probably connected with it. This is located in the lateral gray matter between the anterior and posterior horns and extends from the eighth cervical to the lower edge of the second lumbar segments. Vasomotor and secretory nerve centers are believed to be situated in this tract. The anterior nerve roots contain vasoconstrictor fibers which pass from the cord by means of the rami communicantes in the anterior roots to the sympathetic nerve ganglia. Vasodilator nerves also leave the cord to joint the sympathetic. Fibers pass to the sweat glands in the same way. It can thus be readily seen that by these connections disease of the cord may cause visceral symptoms (Bruce & Pirie, Rev. Neurol. & Psychiat., Jan. 1907, p. 1).

In the lower cervical and upper dorsal regions of the cord is the ciliospinal center (p. 624) from which fibers pass to the sympathetic nerves and supply the vessels of the head and face. It is also connected with the cervical sympathetic which supplies the dilators of the iris (*infra*).

Vasomotor Symptoms.—Vasomotor symptoms consist of abnormal redness or pallor of the skin when there is not sufficient external cause to produce them. Pallor and coolness are due to spasm; redness, heat, and edema, to dilatation of the vessels.

Cyanosis may be due to either dilatation or to spasm, as in Raynaud's disease (p. 800). There may be irregular action, as alternate spasm and dilatation of the vasomotors (vasomotor ataxia), causing local flushing and edemas, as angioneurotic edema, the localized edema of hysteria, Raynaud's disease, erythromelalgia, intermittent claudication, dermographism and urticaria. Apoplectiform attacks (p. 713) and migraine may be due to vasomotor spasm.

Secretory Symptoms.—Secretory symptoms consist of increase or diminution of glandular secretions, as excessive sweating or hyperhidrosis and excessive dryness or anhidrosis. There may be peculiar odors or colors of the secretion. The saliva may be subject to similar disturbances. These disorders are especially liable to occur in derangements of the sympathetic functions and are seen most frequently in the neuroses, as neurasthenia, anxiety neurosis, hysteria.

Disordered function of the cervical sympathetic causes characteristic symptoms that may be of service in the diagnosis of lesions of the cervical cord or nerve roots arising from it (p. 626).

A destructive lesion causes retraction of the eyeball, narrowing of the palpebral fissure, absence of dilatation of the pupil when shaded or cocain is instilled, and in some cases pallor of the face and neck, dryness of the mouth and nose, absence of or diminished sweating in the face and neck on the side of the lesion, and loss of the skin pupillary reflex.

An irritative lesion causes protrusion of the eyeball, widening of the palpebral fissure, dilatation of the pupil, and sometimes flushing of the face and increased sweating.

Vegetative Nervous System.—In this connection mention must be made of the so-called vegetative nervous system. It is a system of efferent fibers which supplies those organs whose action is not under the control of the will, i. e., the glands, organs containing smooth muscle

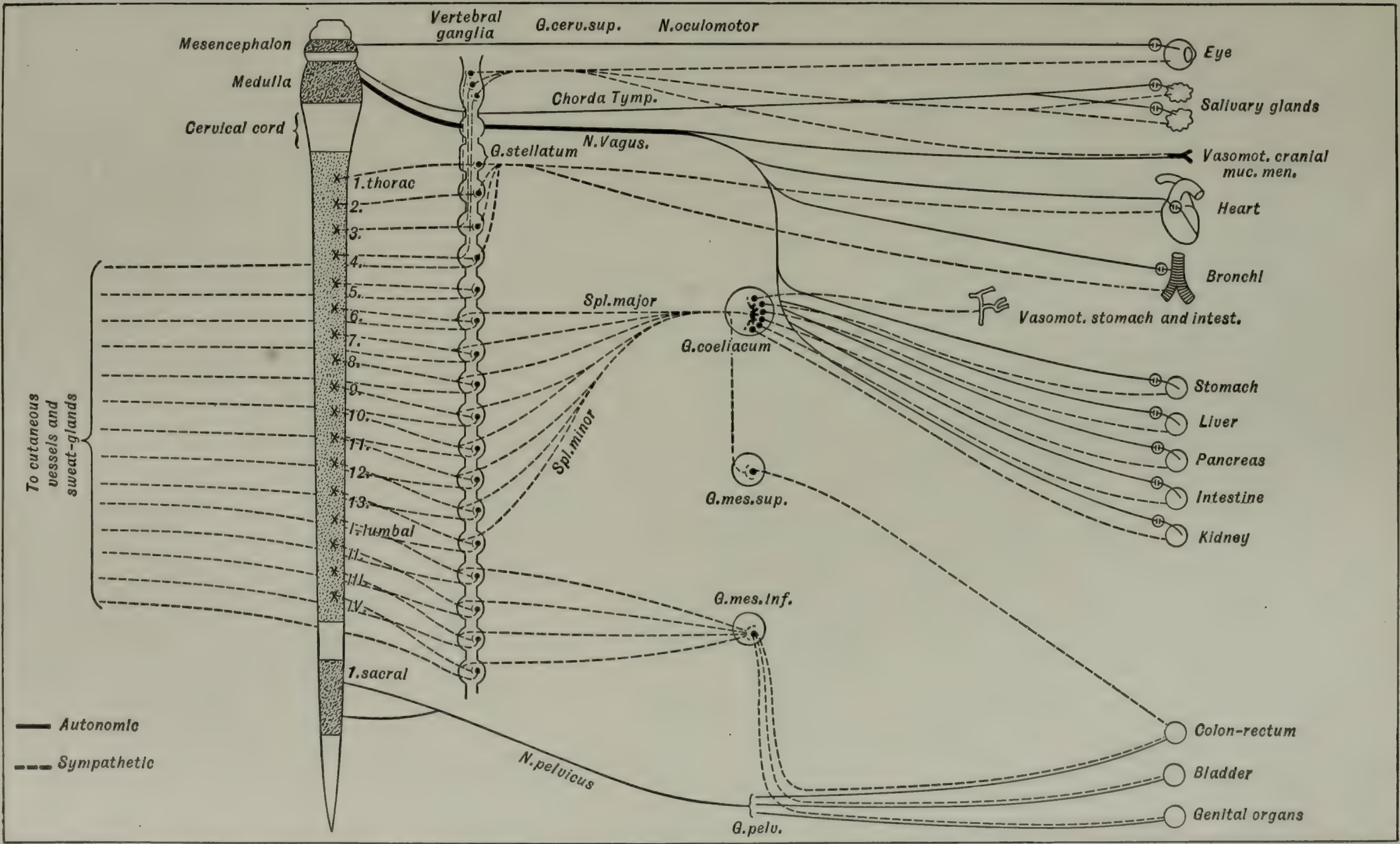


Fig. 126.—Vegetative Nervous System. (After Meyer and Gottlieb's "Pharmacology, Clinical and Experimental," published by J. B. Lippincott Co., Philadelphia.)

fibers, such as the viscera, blood vessels, musculature of the skin, iris, and certain organs containing striated muscle, as the heart, esophagus and penis. It possesses a certain amount of independence of the central nervous system. It is composed of the sympathetic system and some cranial and spinal nerves arising from the sacral segments. The term *autonomic system* is applied to the cranial and spinal nerves (Fig. 126).

This shows very well the nerves constituting the system and the organs which they supply. Abnormal action of these nerves causes characteristic symptoms.

Vagotony or Vagotonia.—One of the most important conditions is that due to overaction of the pneumogastric nerve and sometimes other nerves of the vegetative system and called vagotony or vagotonia. The symptoms of this are many; among them are contracted pupils, accommodation spasm, bronchial asthma, irregular breathing relieved by atropin, bradycardia and cardiac arhythmia, flushing of the face, cold extremities, increased urination and perspiration, dermatographia, absence of the pharyngeal reflex, priapism, increased carbohydrate and fat tolerance before and after taking of epinephrin.

The most important ones are those referable to the gastro-intestinal tract, i. e., spastic constipation, a tender palpable colon, tightly contracted external sphincter, abdominal pain and tenderness which may be mistaken for various lesions of the abdominal organs, as appendicitis, gastric ulcer, gall-bladder disease; pain in the descending colon, more marked either just before or after defecation and which may be excited by cold drinks or rapid eating; attacks of mucous diarrhea; gastric hyperacidity cardiospasm and pylorospasm.

Sympathicotony or Sympathicotonia.—A similar condition of the sympathetic system is known as sympathicotony or sympathicotonia. The symptoms of this are dilated pupils, paralysis of accommodation, dryness of skin and conjunctiva, infrequency of winking, hypoacidity of the stomach, diminished tonus of the intestine, constipation, faulty convergence of the eyes (Möbius' sign), wide palpebral fissure, exophthalmos, tachycardia, high blood pressure, vasomotor spasm (p. 594), urticaria, but not dermatographia, irregular breathing and dyspnea not relieved by atropin, lowered carbohydrate tolerance before and after taking epinephrin and atony of the stomach.

Symptoms of the two conditions may coexist. Certain diagnostic tests are useful, viz.: pilocarpin causes extreme salivation and sweating in vagotonia and none in sympathicotonia. If given 100 gm. of glucose by the mouth and 1 mg. of epinephrin hypodermically about 15 minutes later, no glucose is found in the urine in vagotonia but about 6 gm. are excreted in about five hours in sympathicotonia. Normally a person fasting can take 100 gm. of glucose without causing glycosuria. In vagotonia there is increased tolerance, while in sympathicotonia this amount will cause glycosuria. In addition, ten to fifteen drops of epinephrin (1-1,000) injected hypodermically in one with the latter, cause tachycardia, increased blood pressure, and exophthalmos.

Forced inspiration often causes dilatation and forced expiration causes contraction of the pupil. Pressure on the eyeballs for one minute causes slowing of the pulse in vagotonia. Patients who grow pale when ordinarily the face should flush probably have sympathicotonia.

These cases are frequently mistaken for neurasthenia and hysteria. The differential diagnosis is based on the combination of a number of the symptoms, absence of organic disease, and response to the tests mentioned above.

The fundamental symptom of neurasthenia is abnormal liability to fatigue, of hysteria liability to suggestion (pp. 774-788). This is not the case in the conditions first described. Excellent articles on this subject are those of Spitzig (J. Am. Med. Assn., Jan. 31, 1914, p. 364), Wolfsohn (J. Am. Med. Assn., May 16, 1914, p. 1335), and Held and Gross (J. Am. Med. Assn., Jan. 22, 1916, p. 233).

Trophic Symptoms.—Trophic symptoms consist of atrophy of the muscles; of cutaneous, osseous, and mucous tissues; fragility of the bones; hypertrophy of bones as in acromegaly; joint affections—known as arthropathies or Charcot's joints; and abnormalities of the skin, as scleroderma; ichthyosis; perforating ulcers (usually seen in tabes); herpes due to inflammation of posterior root ganglia or those of cranial nerves; pigmentation; alopecia; painless whitlows with loss of tissue (in syringomyelia); bed sores or decubitus (seen in myelitis and injury to the cord); atrophy of the skin or glossy skin (in neuritis).

Arthropathies usually occur in tabes dorsalis, rarely in syringomyelia. They are characterized by effusion into the joint and separation of the articular surfaces; later there is thickening of the ends of the bones, followed by atrophy, disappearance of the cartilage with undue mobility of the joint (Fig. 170). There is usually absence of pain, redness, and tenderness.

7. Consideration of the Special Senses

(a) Sight

In diseases of the nervous system the sense of sight may be variously affected, the causes of which may be either functional or organic. Examination of the eye is therefore very important and in doubtful cases should be made by an ophthalmologist. A knowledge of the course of the optic tract is essential.

The *visual pathway* commences in the rods and cones of each retina, the deepest layer of which gives origin to the optic nerves. These pass to the chiasm where partial decussation takes place, and the tract from here contains fibers from the nasal side of the retina of the opposite eye and the temporal side of the retina of the eye of the same side (Fig. 127). From the chiasm the tract winds around the cerebral crus, and the fibers pass mostly to the external geniculate body, some to the anterior quadrigeminal body and the posterior part of the optic thalamus or pulvinar. These constitute the *primary optic centers* and are the reflex centers which control the light reflexes of the iris. From these, fibers constituting part of the reflex arc go to the sphincter nucleus in the anterior portion of the third nerve nucleus.

The visual fibers from these centers pass through the posterior part of the posterior limb of the internal capsule and as the radiations of Gratiolet, go to the cuneus above and below the calcarine fissure in the occipital lobe, where are located the cortical visual centers (Figs. 135 and 136). In the calcarine regions of each side therefore are represented the temporal portion of the retina of the same side and the nasal two-thirds of the opposite side.

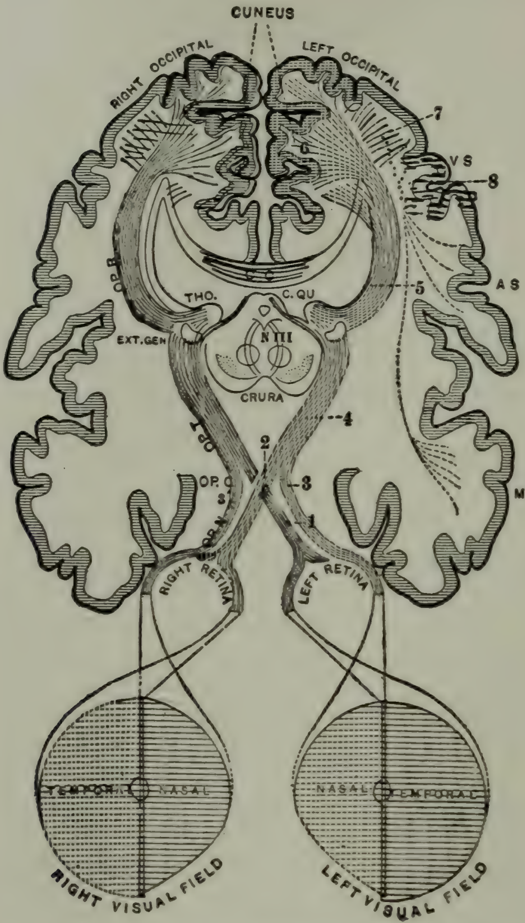


Fig. 127.—Diagram of Visual Paths. (From Vialet, Modified.) OP. N., Optic Nerve; OP. C., Optic Chiasm; OP. T., Optic Tract; OP. R., Optic Radiations; GEN., Geniculate Body; THO., Optic Thalamus; C. QU., Corpora quadrigemina; C. C., Corpus callosum; V. S., Visual Speech Center; A. S., Auditory Speech Center; M. S., Motor Speech Center. A Lesion at 1 Causes Blindness of That Eye; at 2, Bitemporal Hemianopia; at 3, Nasal Hemianopia. Symmetrical Lesions at 3 and 3′ Would Cause Binasal Hemianopia; at 4, Hemianopia of Both Eyes, with Hemianopic Pupillary Inaction; at 5 and 6, Hemianopia of Both Eyes, Pupillary Reflexes Normal; at 7, Amblyopia, especially of Opposite Eye; at 8, on Left Side, Word-blindness. (After Osler.)

Amblyopia.—Amblyopia is a term used to signify dimness of vision. It is usually applied when no gross lesion of the eyeball exists to account for visual failure. It is therefore applied when either functional or organic disease of the retina, optic tract, or cortical centers causes the condition. When complete blindness is so caused it is called *amaurosis*. Diminution of vision in one eye due to lesion of the optic tract of the opposite side is known as *crossed amblyopia*. There is also a slight loss of visual acuity in the other eye. Such cases are rare and indicate that there is a separate region on each side for macular vision. The exact location of this is in dispute. Ferrier believes it to be in the angular gyrus, Henschen and others that it is in the anterior extremity of the calcarine fissure. It is possible that both regions are of importance in subserving this function.

Functional amblyopia or *amaurosis* may be caused by hysteria, neurasthenia, anemia, and reflex irritations, as decayed teeth. They are also caused by various poisons, notably tobacco, alcohol, lead, quinin, salicylic acid, and in diabetes, uremia, and infectious diseases. In such cases the lesion is probably a retrobulbar neuritis.

RETINAL HYPERESTHESIA.—Retinal hyperesthesia is seen in meningitis and hysteria.

OPTIC NEURITIS.—This is a frequent symptom in certain diseases of the nervous system. The form so caused is usually that in which there are lesions of the intra-ocular end of the nerve (retrobulbar neuritis is mentioned above). It may be found in meningitis, syphilis, acute infectious diseases, poisoning by lead, alcohol, in rheumatism and often after exposure to cold.

CHOKED DISK OR PAPILLEDEMA.—The condition of the optic nerve of greatest importance in neurology is the *choked disk* or *papilledema*. This consists of swelling of the nerve head and its projection into the interior of the eye. It is probably not a true inflammation, but is due to edema caused by compression of the vessels as they pass through the lamina cribrosa or nerve (Cushing and Bordley). Brain tumor is the most frequent cause; anything, however, causing an increase in intercranial pressure, as cerebral hemorrhage or abscess, may produce it.

A condition resembling it may also be caused by thrombosis of the cavernous sinus, inflammation of the accessory sinuses (frontal, alveolar), nephritis, anemia, and disease of the orbital region. Both optic neuritis and papilledema may exist without any impairment of sight.

OPTIC ATROPHY.—Optic atrophy may be primary, and may occur in tabes dorsalis, paresis, and multiple sclerosis. There is also a hereditary form (amaurotic family idiocy); a secondary form which results from pressure more or less directly applied to the optic chiasm or tracts, as in pituitary disease; and a consecutive form which is a secondary consequence of optic neuritis or papilledema. The symptoms in general are failure of vision, contraction of the visual fields, and altered perception of colors.

Hemianopsia.—Hemianopsia denotes blindness of one-half the visual field, and is due to a lesion of either the chiasm, tract, or centers. If the dividing line is vertical it is termed *vertical hemianopsia,* and *horizontal hemianopsia* if the line is horizontal. The former is the more important. It may be *bitemporal,* in which both temporal fields are wanting. This can be caused only by a lesion involving the crossed fibers of both optic tracts in the middle of the chiasm (Fig. 127); it may be *binasal,* in which both nasal fields are absent. This is caused by a lesion on both sides of the chiasm or two lesions, one on the opposite side of each optic nerve (Fig. 127); and *homonymous,* the most common, in which the temporal of one eye and the nasal field of the other are wanting (Fig. 127). If both right halves are wanting it is *right homonymous hemianopsia,* and vice versa. The loss of function is always in the opposite side of the retina from the blind field and the lesion is on the opposite side also (Fig. 127). It must be in the optic tract posterior to the chiasm. If posterior to the primary optic centers (p. 598) Wernicke's sign (p. 591) is absent.

Homonymous hemianopsia may be *complete,* that is, the entire half field with the exception of the small area at the center (macular vision) where the visual area extends a little to the far side of the dividing line, is lost. It may be *absolute,* all forms of vision, viz., perception of light, form and color, being lost. It may be *relative,* that is, light sense is preserved, but one or both of the other forms (*supra*) is lost.

Relative hemianopsia is always due to a cortical lesion (Cuneus). Cortical lesions, however, may also cause the absolute form. If due to a cortical lesion there is always contraction of the preserved half field. A lesion above the calcarine fissure causes blindness in the lower quadrant, one below the fissure in the upper quadrant. Homonymous hemianopsia is rarely due to functional causes, but may occur as a symptom of migraine and hysteria. In such cases it is usually transient.

Dyschromatopsia.—Visual fields may be contracted in disease of the optic nerve (atrophy, papilledema, neuritis), and in functional diseases (hysteria, neurasthenia). The normal order of color perception is often interfered with, especially in hysteria, sometimes as a general symptom of brain tumor. Normally the largest field is for blue, then red, and lastly green. This order may be reversed in various ways (Fig. 128). The symptom is known as dyschromatopsia.

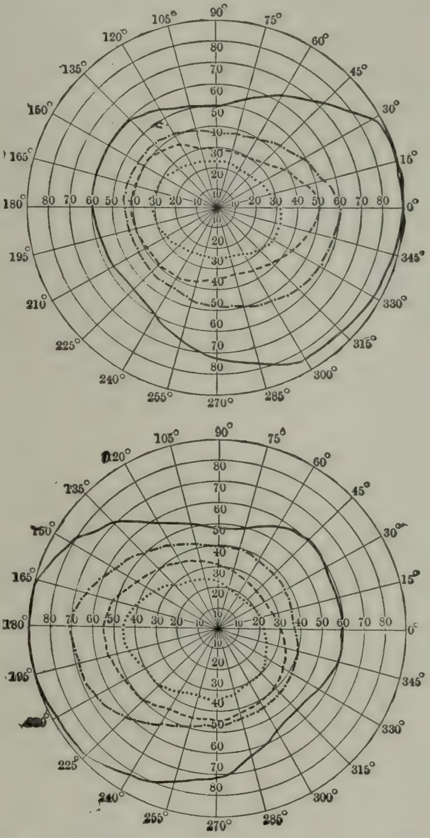
Achromatopsia.—Achromatopsia is inability to recognize colors (color blindness).

Examination. — Tests for the pupillary reflexes are detailed on page 591.

Contraction of the fields and hemianopsia may be roughly tested by placing the patient three or four feet away in front of you with one eye covered. Cause him to look steadily at the end of your nose. A white object or one or two fingers is then brought from different points of the periphery toward your nose and the patient told to say when he sees it. If the fingers are used he may be asked if they are moving or still or how many are seen. Another plan, if patients are somewhat stuporous, is to bring an article of food or something that the patient would be interested in, into what should be the field of vision; if he sees it he will instinctively turn the eyes toward it. The presence or absence of *exophthalmos* should be noted.

Diplopia or *double vision*, by which is meant seeing two objects when looking at one, is due, when persistent, to weakness of one or more of the ocular muscles supplied by the third, fourth or sixth nerves (*vide*). It may be an early symptom of cerebral syphilis, tabes dorsalis, and multiple sclerosis. It must be remembered that weakness of extra-ocular muscles causing squint may be congenital and not dependent on disease of the nervous system.

The muscles affected may be roughly determined by these tests: If the weakness is marked, squint will be noticed. If not sufficient to cause this the patient is instructed to follow first with one eye (the other being covered) and then with both, without turning the head, the finger or lighted candle moved by the examiner in different directions. If he cannot move the right eye to the right, the right external rectus is paralyzed, and so on.



—.—. =red. - - - -=blue. . . . . =green

Fig. 128.—Diagram of Fields of Vision in a Case of Hysteria, Showing Normal Form Fields and Reversal of the Red and Blue Fields, the Red Field Being Largest in Extent. (After Mitchell and de Schweinitz, from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

If a more delicate test is desired he is instructed to fix the moving finger of the examiner or candle with both eyes, one of which may be covered with a colored glass to differentiate the images and as soon as two images are seen it must be stated. The diplopia increases as the object is moved toward the side of the paralyzed muscle. The image seen by the sound eye is the *true image*, that by the affected eye the *false image*. If the latter is on the same side as the eye by which it is seen, i. e., if the right image disappears when the right eye is closed, it is called *homony-*

mous diplopia, if it does not it is *crossed diplopia.* In other words, when the prolonged axes of the eyes would cross, the double vision is not crossed (Gowers). Convergent strabismus therefore is accompanied by homonymous, and divergent strabismus by crossed diplopia.

Monocular diplopia, i. e., seeing two images with the eye, occurs sometimes in hysteria.

Noting the presence or absence of nystagmus is important (p. 583). Sometimes when not apparent with the eyes at rest it may be brought out by causing the patient to move the eyes, as in testing the muscles (p. 601). It is important not to mistake coarse, jerky movements that may occur when some of the muscles which move the eyeball are weak, for true nystagmus. If the nystagmoid movements are due to weakness it will be shown by applying the tests for such weakness (*supra*).

Examination of the pupils is described on page 591.

(b) Hearing

The acuity of hearing can be roughly determined by blindfolding the patient and covering one ear. A watch is held some distance from the other ear and gradually brought toward it until the patient indicates he hears it. The normal distance is from two to four feet, according to the loudness of the tick. In order to determine if there is trouble in the internal ear, when bone conduction will be interfered with, a large tuning fork (note C) is struck and held first at varying distances from the ear and then placed either on top of the head or over the mastoid process. If the sound is heard better when placed before the ear, aërial conduction is better than bone conduction, and vice versa. If when placed on top of the head the sound is heard better in the non-affected ear the internal ear is diseased. If it is heard better in the affected ear the trouble is in the middle ear or auditory canal (Rinne's and Weber's tests).

The caloric, electrical, and tuning tests of Bárány are now much used to determine disease of the internal ear, and of certain parts of the brain (p. 584).

(c) Smell

Care should be taken in testing this to use odors which are not irritating, as peppermint, asafetida, or toilet extracts. These are approached to one nostril, then the other, the one not being tested being plugged. The possibility of catarrhal trouble causing loss of smell must be considered.

(d) Taste

The cortical center for taste is probably in the region of the uncinate gyrus (Fig. 136). The chorda tympani, which runs through the facial canal with the seventh nerve and then joins the lingual branch of the fifth, supplies the anterior two-thirds, while the glossopharyngeal, or ninth nerve, supplies the posterior part of the tongue with taste fibers. It is probable that the chorda tympani, by means of the intermediary nerve of Wrisberg, is connected with the ninth nerve (p. 648).

Pure taste sensations are bitter, sweet, sour and salt. If associated with an odor, it is called a flavor; anything which causes loss of smell interferes with the appreciation of flavors.

Disease of the seventh nerve within the facial canal is the most common cause of loss of taste (p. 643). It may also be present on one side in the hemianesthesia of hysteria (p. 577). Hallucinations of taste may occur in insanity and in disease of the uncinate gyrus (p. 768) and may be the aura of epileptic attacks as may also sensations of smell. Perversions (parageusia) and increased sensitiveness (hypergeusia) may occur in hysteria.

Examination.—To test the sense of taste we use common salt; a sweet taste, sugar; a sour taste, vinegar; and a bitter taste, a solution of quinin or strychnin. The bitter should be used last.

The patient keeps the tongue protruded and one of the solutions is painted on the suspected side with a camel's hair brush or piece of absorbent cotton wound about an applicator, first anteriorly then posteriorly. The substance must be rubbed in well and a few seconds allowed for the patient to recognize the substance. The tongue must be kept protruded during the entire test and the patient can indicate if he recognizes the taste by pointing to the name of it which has been written on a piece of paper with those of the other substances used.

(e) Speech

Disturbances of speech are of importance in neurology. They are discussed on page 614.

7. Electricity as a Means of Diagnosis

Both the constant, or galvanic, and the faradic, or induced, currents are of much service in neurological diagnosis. In certain diseases of the nervous system the muscles and nerves when stimulated by these forms of electricity act differently than do normal muscles and nerves.

The *electrical irritability* may differ in two ways—either as a *quantitative change,* meaning either a diminished or increased irritability so that either a weaker or stronger current is needed to cause a response than is the case in the normal person, or a *qualitative change,* meaning a change in the character of the contraction.

Quantitative Changes.—An increased excitability occurs in the early stages of the reaction of degeneration. It is also part of the phenomena of the myotonic reaction (*infra*) in which there are also qualitative changes. As a pure quantitative change it is best seen in tetany.

In this the motor nerves respond to a very weak constant current (one milliampere or less). A slight increase will cause a tetanic contraction. The ulnar nerves are especially excitable. This is known as *Erb's symptom.* To elicit it place a large electrode (the positive pole or anode) at some indifferent point and place a small one (the negative pole or cathode) over the nerve (See Motor Points). Beginning at zero, very gradually increase the current strength while opening and closing the circuit, and noticing when the muscles supplied by the nerve being tested contract. In tetany this will happen with a much weaker current than normal. Thomas found in one case that when he placed the cathode over the nerve, fibrillary contractions were noticed in the muscles with a current much less than one milliampere in strength. As the strength was gradually increased the muscles were then tetanized. He called this *catelectrotonus tetanus.*

If in a case of tetany the electrode is placed over a sensory nerve, tingling sensations may be felt in the distribution of the nerve with very weak currents (Hoffman's symptom).

Diminished excitability is found in the reaction of degeneration (*infra*), in mild cases of peripheral motor neuron lesion (p. 563) and in paralysis of long standing due to a central motor neuron lesion (p. 610).

To test this the electrodes are placed over the nerves and motor points of the muscles, and the current increased as described above. Both the constant and faradic currents should be tried. If the paralysis is unilateral it is well to find the weakest current that will cause contraction in the normal side first. If bilateral the examiner's own muscles may be used as a control, if necessary.

Qualitative Changes.—The only pure qualitative change of importance is the *myasthenic reaction,* which is a symptom of myasthenia gravis. It shows itself by the rapid exhaustion of a muscle when stimulated with the faradic current. After a brief rest the muscle will react normally, to again soon lose the power of contraction if the stimulus is continued.

It is dangerous to test the muscles of respiration, as failure of respiration may occur. In making the test a small electrode should be placed over the nerves and motor points and a rapidly interrupted faradic current used. Contraction should be continued for at least five minutes.

Sometimes, as in a case examined by Mosher, there are irregular, jerky contractions without complete relaxation between them.

Quantitative and qualitative changes are found in the *reaction of degeneration* designated by the symbols, De R, R D or D R, and the *myotonic reaction.*

The D R, as defined by Erb, is "an entire cycle of quantitative-qualitative changes of irritability, which occurs in the nerves and muscles under certain pathological conditions and presents intimate relations to certain histological degenerative changes occurring in these structures. It is characterized in the main by the diminution and loss of the faradic and galvanic irritability of the affected nerve and the faradic irritability of the muscles; while the galvanic irritability of the muscles persists, it sometimes is considerably increased and is always changed qualitatively in a definite manner." These changes may not all be present, in which case we speak of a *partial or incomplete reaction of degeneration.* The qualitative changes are a slow, tetanic contraction of the muscles as compared with the sharp, quick contraction of the normal (*the modal change*) and the so-called *serial change,* which consists of a reversal of the normal formula of contraction.

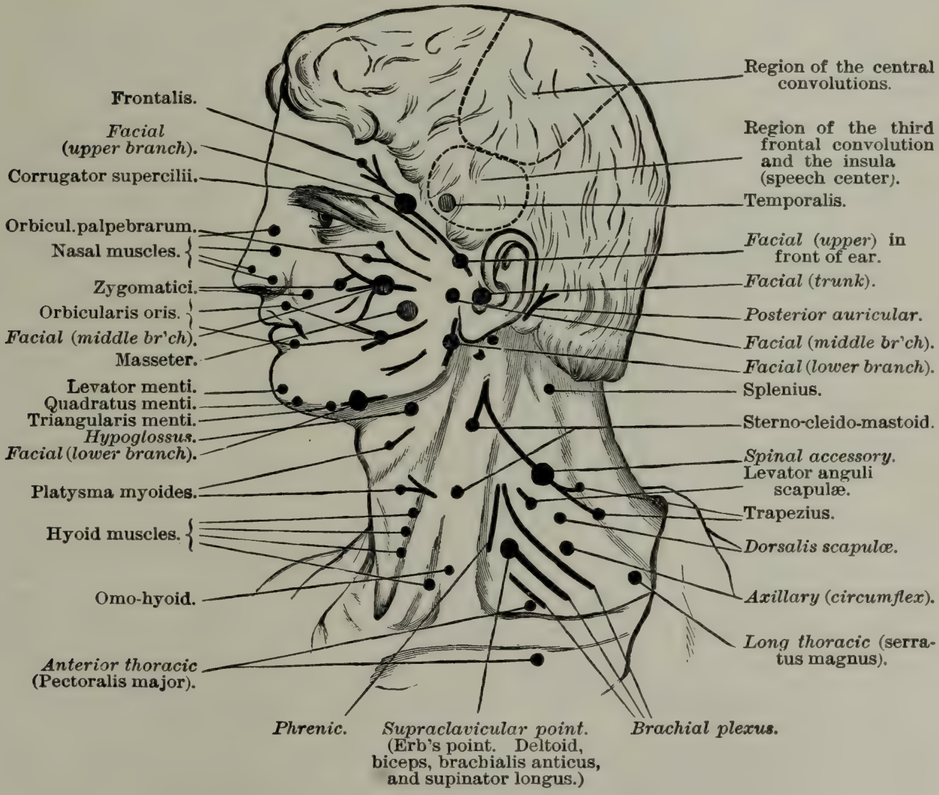


Fig. 129.—Motor Points of Muscles and Peripheral Nerves. (After Erb.)

Normally the weakest constant current which will cause a muscular contraction is that in which the cathode is made the exciting pole and the circuit closed. This is known as a *cathodal closure contraction* (C Cl C). If the current is made stronger we get contraction, when the anode is made the exciting pole and the circuit closed or opened (An Cl C and An O C), indicated by the formula C Cl C″, An Cl C′, and O C, C O C. In the D R this is reversed, the contraction occurring either first, when the anode is the exciting pole or equally with the cathode. These changes can be indicated by the formulae:

An Cl C   C Cl C   or An Cl C=C Cl C.
An O C   C Cl C   or An O C=C Cl C.

C O C is very rarely seen.

In most cases there is a marked increased excitability of the muscles to the constant current lasting from three to eight weeks, followed by a

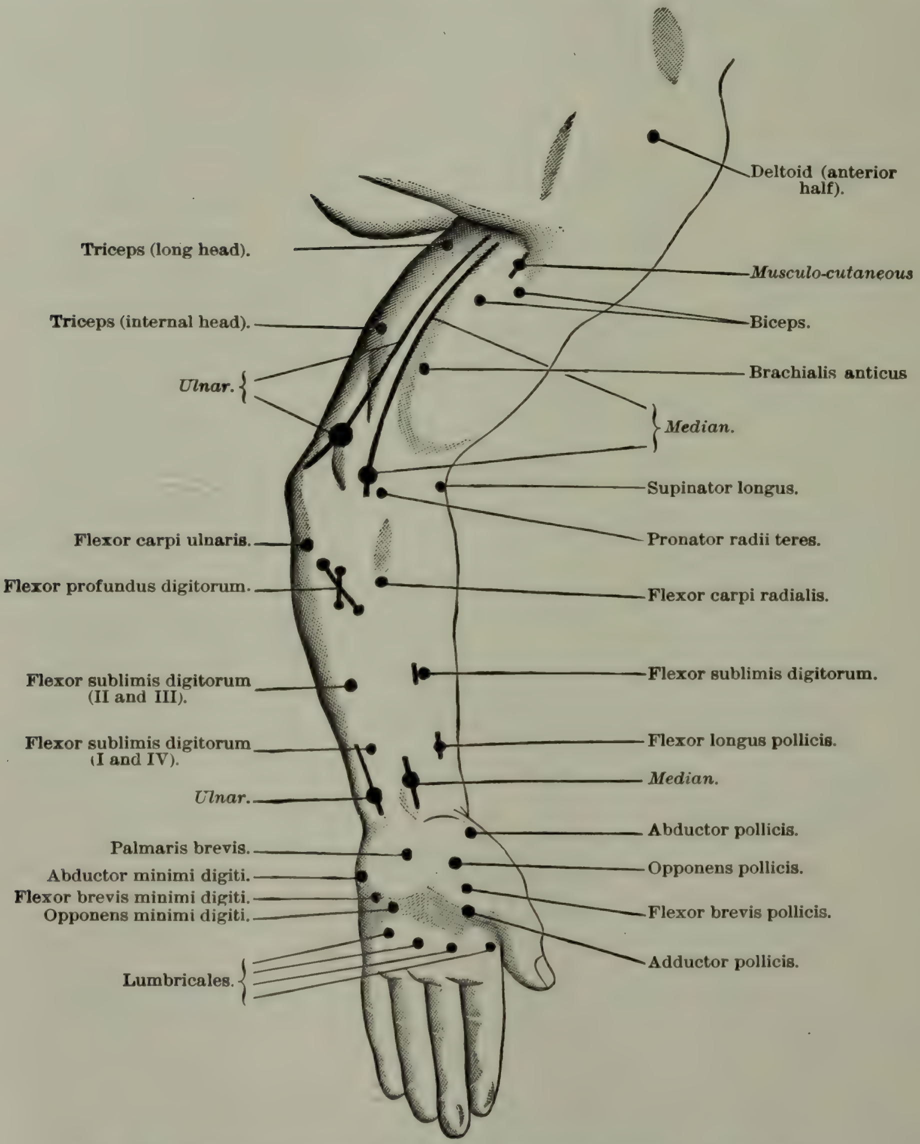


Fig. 130.—Motor Points of Muscles and Peripheral Nerves. (After Erb.)

diminished excitability. The partial reaction may consist of different variations of the above, the constant changes being diminished faradic irritability and the modal change.

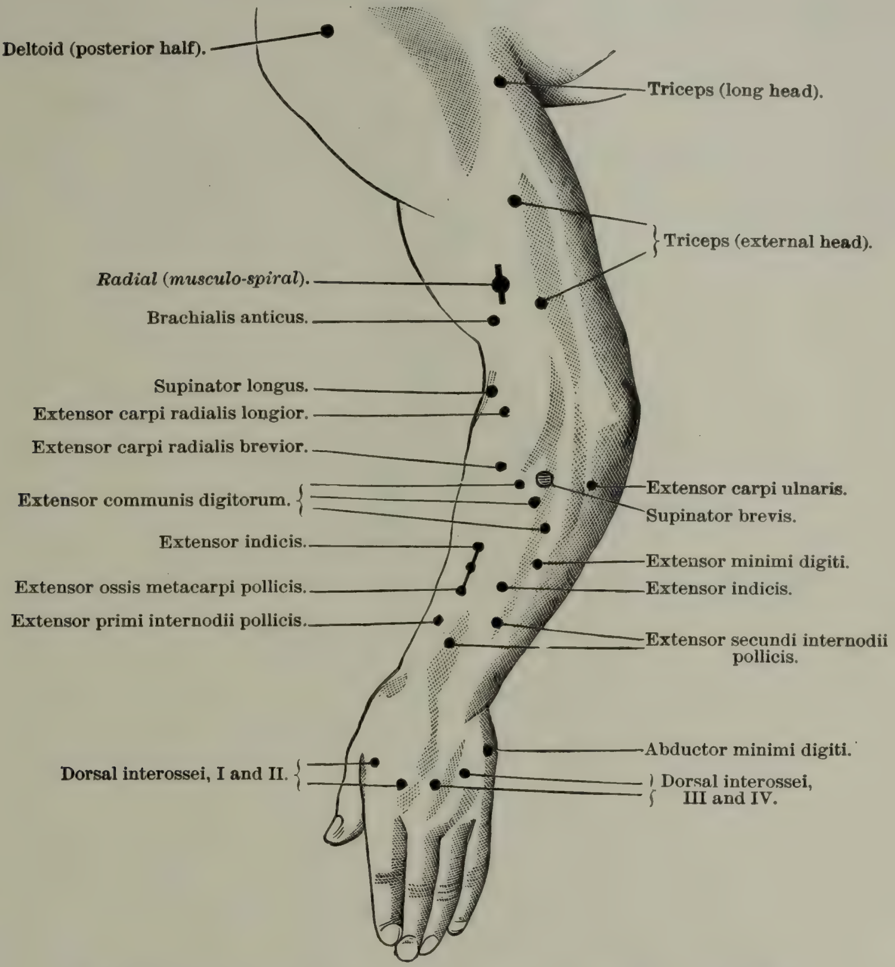

Deltoid (posterior half).
Triceps (long head).
Triceps (external head).
Radial (musculo-spiral).
Brachialis anticus.
Supinator longus.
Extensor carpi radialis longior.
Extensor carpi radialis brevior.
Extensor communis digitorum.
Extensor carpi ulnaris.
Supinator brevis.
Extensor indicis.
Extensor minimi digiti.
Extensor ossis metacarpi pollicis.
Extensor indicis.
Extensor primi internodii pollicis.
Extensor secundi internodii pollicis.
Abductor minimi digiti.
Dorsal interossei, I and II.
Dorsal interossei, III and IV.

Fig. 131.—Motor Points of Muscles and Peripheral Nerves. (After Erb.)

In testing for the presence of D R, it is important to place a large, moist electrode at a point equidistant from the limbs of each side of the body, as the sternum or sacrum. A small electrode is used for the active one. The nerves should be tested first, this being done by placing this electrode first on the corresponding nerve of the normal side if the paralysis is unilateral, and then over the nerve to be tested (See Figs. 129-134) and in each case beginning at zero, gradually increasing the current strength until contraction occurs. This should be done with both currents and the amount of coil necessary with the faradic and the number of milliamperes or cells needed with the constant current noted. The electrode is then placed over the motor points of the muscles (*infra*) and the same process repeated. Here the character of the contraction must be closely watched (modal change) and if it first appears when the anode is the exciting pole or the cathode or if they are about equal. In bilateral paralysis, the corresponding muscle and nerves of a normal person may be used as controls if necessary. While being tested the muscles must be as relaxed as possible.

Motor Points. These are points upon the surface of the body from which the respective muscles can be excited most easily. They correspond to the points of entrance into the muscle of their motor nerve branches. These have been mapped out in Figures 129 to 134.

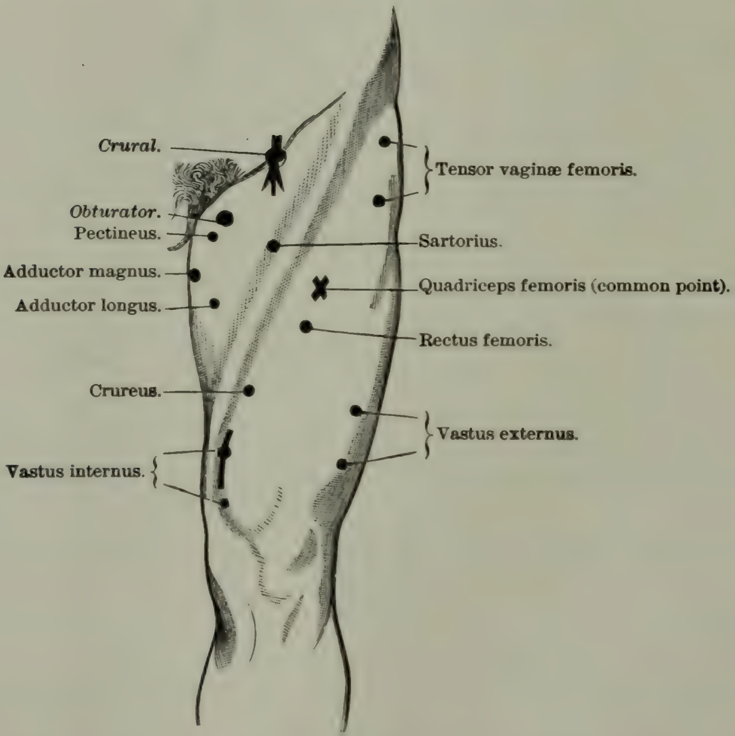


Fig. 132.—Motor Points of Muscles and Peripheral Nerves. (After Erb.)

The D R occurs only in motor paralysis due to peripheral neuron lesion; it is therefore a distinguishing symptom from central neuron paralysis. It is also of service in prognosis. As Erb says, "Other things being equal, the lesion is serious, probable duration of the disease longer and the definite prospect of a cure more remote in proportion as the D R is developed and complete, and in proportion to the stage which it has reached."

Myotonic Reaction.—The myotonic reaction is only found in myotonia congenita or Thomsen's disease (p. 530). It consists of a normal reaction of the nerves to both faradic and constant currents. The muscles when directly stimulated with a minimal faradic current react normally, but if the current is increased a tetanic contraction appears rapidly which persists for some time after the stimulus is removed. Strong faradic currents sometimes also cause irregular, undulating contractions. If stimulated with the constant current only closure contractions are produced, but An Cl C=C Cl C and they are slow, tonic and prolonged, like those produced with the faradic current. As they are produced by both currents they differ from D R.

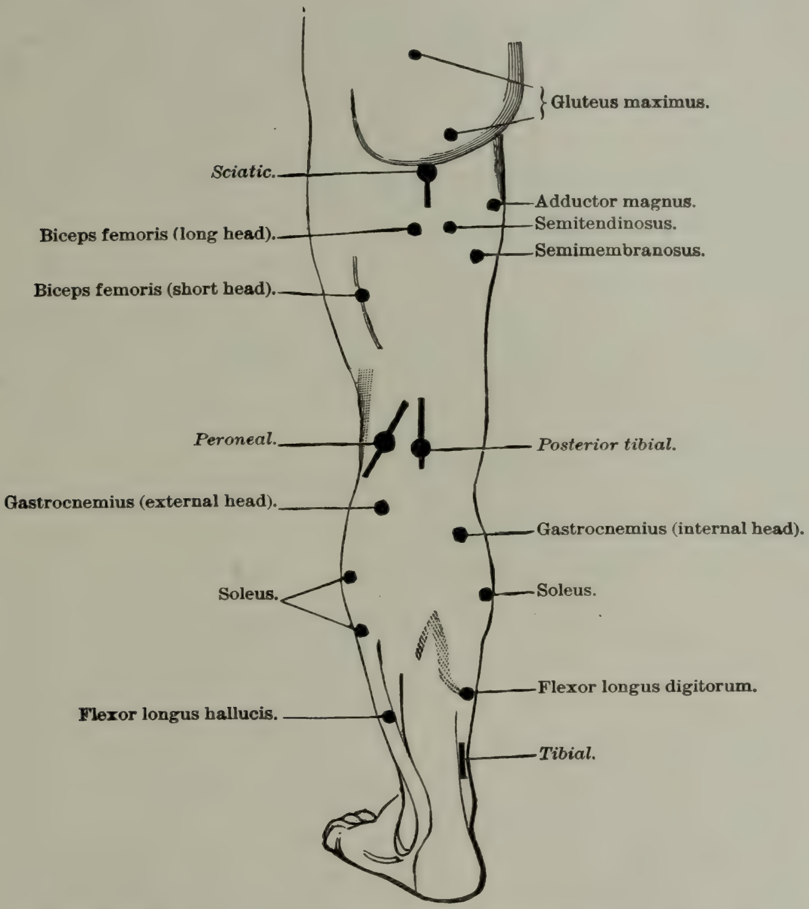


Fig. 133.—Motor Points of Muscles and Peripheral Nerves. (After Erb.)

What are known as Erb's waves are sometimes seen. These consist of wavelike movements of the muscle passing from the cathode to the anode when a strong uninterrupted constant current passes through the muscle. The methods of examination are similar to those described for D R. The following table from Potts' "Electricity—Medical and Surgical" shows the electrical changes useful in diagnosis.

Table Showing the Connection Between Pathological States of the Motor Tract and Muscles and Their Electrodiagnostic Symptoms (Functional Disorders Are Included)

Seat of Lesion and the Disease	Electrical Reactions
Brain cortex and the pyramidal tracts (central neuron), Cu-Py, Fig. 114, viz.: Cerebral apoplexy and the resultant paralysis; tumor; abscess; transverse or compression myelitis (muscles supplied by nerves arising from segments below the seat of the lesion); lateral sclerosis; hysterical paralysis	Normal usually. If paralysis is of long duration, sometimes slight diminution of excitability
Pyramidal tracts and the cells of the anterior horns of the cord (central and peripheral neurons). Py-Sc, Fig. 114, amyotrophic lateral sclerosis	In non-atrophied muscles—normal. In atrophied muscles—either quantitative decrease or DeR (usually partial)
Cells of the anterior horns of the cord and bulbar motor nuclei (peripheral neuron) Fig. 114	
a, acute poliomyelitis; transverse myelitis, tumor, hemorrhage, localized meningitis, syringomyelia (muscles supplied by nerves from the affected segments when the gray matter of the cord is involved); glossolabiolaryngeal paralysis	*a*, various degrees of DeR, in acute poliomyelitis most often complete
b, progressive muscular atrophy	*b*, quantitative decrease in early stages. Various degrees of DeR in the later. This may also apply to syringomyelia, localized meningitis, and glossolabiolaryngeal paralysis
c, myasthenia gravis	*c*, myasthenic reaction
Peripheral nerves (peripheral neuron), PU, Fig. 114; neuritis from various causes, as rheumatic, traumatic, or toxic; progressive neuritic muscular atrophy	Quantitative decrease in mild cases. Various degrees of DeR in the more severe ones
Muscles. M, Fig. 114	
a, myopathies or dystrophies	*a*, normal or quantitative decrease
b, myotonia congenita	*b*, myotonic reaction
c, tetany	*c*, quantitative increase

The *constant current* may be used to test the *sense of taste,* by placing two very small pointed electrodes or the metal ends of the conducting cords upon the tongue and using a very weak current. A metallic taste is noticed if the sense is intact. The constant current may be used to determine *disease of the auditory nerve.* This is done by placing the active electrode in front of the ear, the indifferent at some remote point. As the current, which must not be interrupted, passes, nystagmus is caused. Normally when the anode is used the movement is away from the electrode and toward it if the cathode is used. If the nerve is diseased, less anodal milliamperes will be required to cause nystagmus than cathodal. It requires some skill to make these observations.

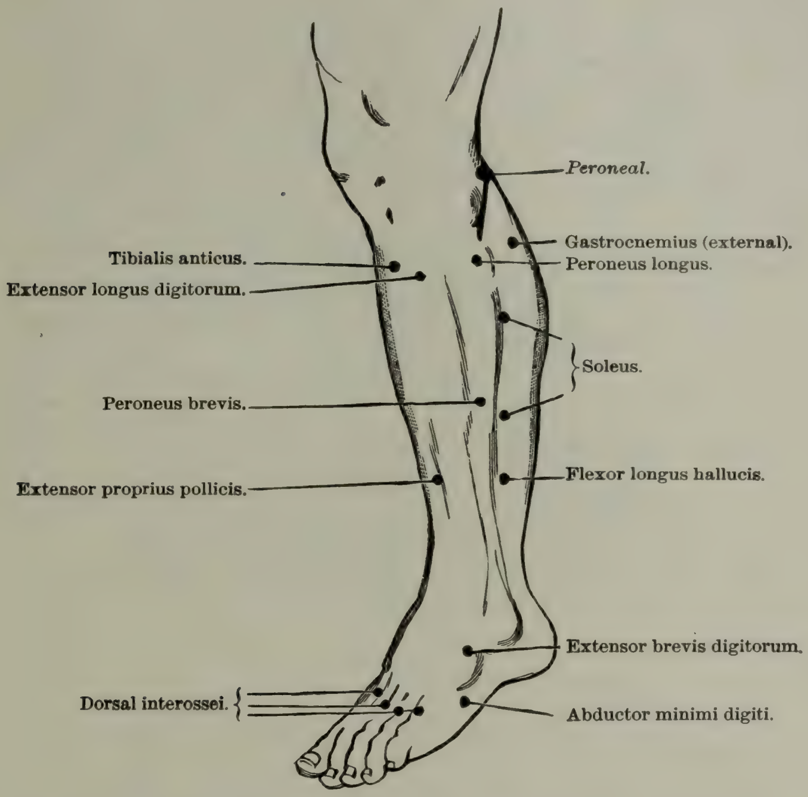


Fig. 134.—Motor Points of Muscles and Peripheral Nerves. (After Erb.)

Examination of the cerebrospinal fluid obtained by *lumbar puncture* is of considerable importance in both the diagnosis and prognosis of diseases of the nervous system due to syphilis. This has been described on pages 748, 751, 754.

9. Cerebral Localization

In determining the locality of a lesion in the brain it must be remembered that symptoms referable to the centers in which the lesion is situated may be not the only ones present; others, either destructive or irritative (pp. 557, 562), are usually present, due either to compression or irritation of neighboring centers or tracts. If the lesion increases the size of the brain or takes up part of the skull cavity, the brain may be considerably distorted, and this also may cause symptoms due to disturbance of the function of parts remote from the lesion. Cerebral motor centers represent movements and not individual muscles; therefore several muscles are represented in a motor center.

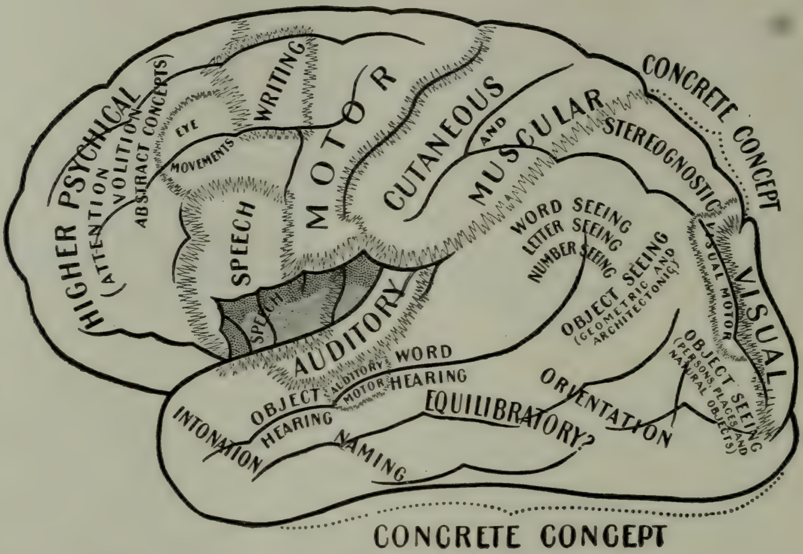


Fig. 135.—Side View of Human Brain, Showing Localization of Functions. (After Charles K. Mills; from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

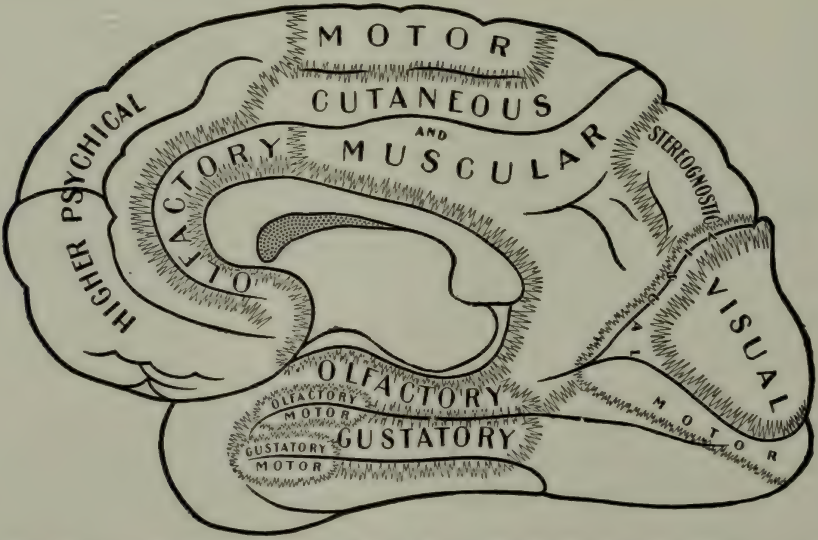


Fig. 136.—View of the Mesial Surface of the Human Brain, Showing Localization of Functions. (After Charles K. Mills; from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

Figures 135, 136 and 137 show the different cortical centers of the cerebrum. Figure 137 shows the subdivisions of the motor cortex. In general it may be said that a destructive lesion causes lessening or loss of function of a center (paralysis), while an irritative one causes increased function.

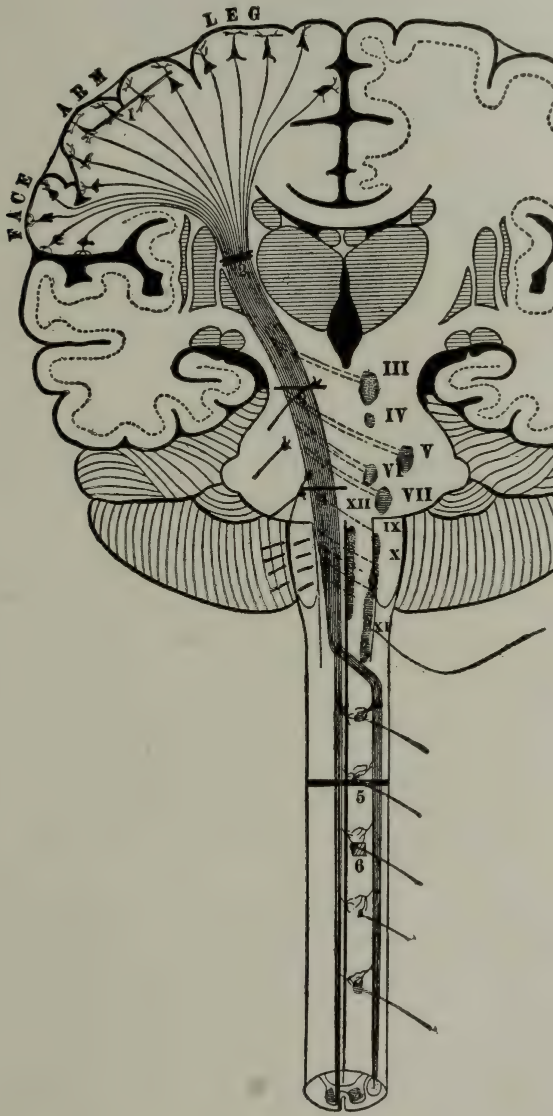


Fig. 137.—Diagram of Motor Path from Left Brain. A Lesion at 1 Would Cause Upper Segment Paralysis in the Arm of the Opposite Side—Cerebral Monoplegia; at 2, Upper Segment Paralysis of the Whole Opposite Side of the Body—Hemiplegia; at 3, Upper Segment Paralysis of the Opposite Face, Arm, and Leg, and Lower Segment Paralysis of the Eye Muscles on the Same Side—Crossed Paralysis; at 4, Upper Segment Paralysis of Opposite Arm and Leg, and Lower Segment Paralysis of the Face and the External Rectus on the Same Side—Crossed Paralysis; at 5, Upper Segment Paralysis of All Muscles below Lesion, and Lower Segment Paralysis of Muscles Represented at Level of Lesion—Spinal Paraplegia; at 6, Lower Segment Paralysis of Muscles Localized at Seat of Lesion—Anterior Poliomyelitis. (Van Gehuchten, Modified.)

Jacksonian Epilepsy.—In the case of centers for special senses this may be manifested as hallucinations. If a motor center there are periodical attacks of clonic spasm in the muscles supplied by the particular center, which may or may not spread to other muscles and is unattended with loss of consciousness unless, as is rarely the case, the convulsion after several minutes becomes general, when consciousness may be lost. These spells are known as Jacksonian epilepsy.

The *prefrontal region* or that part of the frontal lobes in front of the ascending and third frontal convolutions is the seat of the higher mental processes, as memory, attention, judgment, reasoning, etc. It is thought by many that these are principally located in the left side in right-handed people, and vice versa. It is probable that in this region centers for muscle tone are located (p. 558). Lesions of the first and second frontal convolutions of the left side have caused apraxia (p. 617) and "tonic innervation" (p. 559).

The centers for *voluntary motor impulses* are located in the entire length of the ascending frontal convolution, partly in the second frontal and in the paracentral lobules, those for leg movements being in the latter and upper part of the ascending frontal.

A cerebral lesion causing *monoplegia* is always in or near the cortex, as it is only here that there is space enough for a lesion to involve the centers for one limb without involving the others (Fig. 137).

Muscles that habitually act together, as those of respiration, swallowing, expression, are represented on both sides of the brain and a one-sided cerebral lesion does not usually cause their permanent paralysis, as the other side of the brain takes up the work (pp. 642, 708).

Sensory impressions are received in the ascending parietal convolutions and parietal lobe. The *superior parietal lobule* contains the centers for *muscle and the stereognostic sense* (p. 578). Lesions of the left side may cause apraxia.

The centers for taste and smell are located in the uncinate region. *Hearing* is probably located in the first temporal convolutions, although the centers of both sides must be destroyed to cause any loss of hearing. *Memories for words* and *special sounds* are in the first and second temporal convolutions of the left side in right-handed people.

The *occipital lobe* contains the *visual centers* (Fig. 135 and 136) which are described on page 597.

Aphasia

Most important are those regions of the cortex which have to do with speech and related functions, as writing and reading. A lesion of any or all of these causes a combination of symptoms known as aphasia. By this we mean the partial or complete loss of either the power of expression or of comprehension, or both combined, of any of the usual signs of language, not dependent upon lesions of the peripheral nerves or muscles concerned in speech, but upon lesions of the cortical centers or tracts connecting them.

To appreciate the functions of these centers some knowledge of the genesis of speech is essential. The child learns to understand words before he can utter them. Through the different senses he perceives the different characteristics of an object. These percepts are stored away in the brain, and eventually the child learns to associate the name that he hears applied to the object with that object. The memory of the sound of this word is stored in the *center for word memories* (which in right-handed people is located in the *first temporal convolution* of the left side, and vice versa (Fig. 138). Whenever he hears this word used the different characteristics of the object are brought into consciousness by stimulation

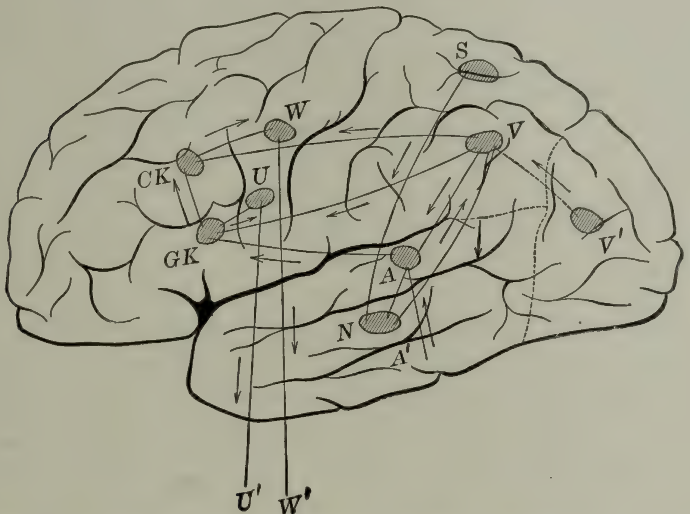


Fig. 138.—Diagram Showing Probable Pathways of Nervous Impulses Concerned in Speech and Writing:[1] A, Center for Auditory Word Memories in First Temporal Convolution; V, Center for Visual Word Memories in Angular Gyrus; GK, Glossokinesthetic Center (Bastian), or Psychomotor Center, at Foot of Third Convolution; U, Centers for Muscles Involved in Articulation of Foot at Ascending Frontal Convolution; CK, Probable Centers for Memory of Muscular Movements Involved in Writing, Cheirokinesthetic Center of Bastian; W, Centers for Controlling Muscles of Arm and Hand in Ascending Frontal Convolution; A-A', Tract from Cortex of Temporal Lobe (Auditory Centers) to Center for Word Memories; V-V', Tract from Cuneus to Center for Visual Memories; W-W', Tract from Arm and Hand Centers to Cells in Anterior Horns of Cord and Peripheral Nerves Controlling These Muscles (Pyramidal Tract); U-U', Tract from Centers for Muscles of Articulation to Centers in Pons and Medulla and Nerves Controlling Those Muscles (Pyramidal Tract). In Speaking, Impulses Travel from A-GK-U-U'; in Reading Aloud, from V-A-GK-U-U'; in Silent Reading, from V-A-GK; in Writing Spontaneously, V-A-GK-CK-W-W'; in Writing from Dictation, A-V-GK-CK-W-W'; in Copying, V-CK-W-W'; N, Naming Center; S, Stereognostic Center in Parietal Lobe. (After Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

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[1] In Right-handed Persons These Centers and Tracts Are Situated in the Left Side of the Brain; in Left-handed Persons the Opposite Is the Case.

of the different centers where the memories of these characteristics are stored (visual, stereognostic, etc.) (Fig. 138). Gradually the child learns to make and coördinate the muscular movements of the lips, tongue, larynx, necessary to pronounce the word. The *memory of these movements* is located in right-handed people in the third left frontal convolution (Fig. 138), and vice versa. This is variously known as *Broca's convolution, psychomotor center, glossokinesthetic center of Bastian.*

The exciting of one of these centers (auditory, visual, stereognostic, etc.) excites the others and if, for instance, we hear a bell ring, the other characteristics of a bell are brought into consciousness, so that in the mind's eye we can see it, feel it, etc. The proper word designating this image also comes into consciousness by stimulation of the center for word memories. If it is desired to pronounce the word the *motor speech center* (third frontal) is stimulated by an impulse from the *first temporal* (*auditory memories*), and the various coördinate muscular movements necessary to pronounce the word are then made.

The motor speech center can also be excited without the word being pronounced audibly. This is constantly done in silent thinking and reading, the sounds of words being mentally recalled without visible movements of the muscles being made. This is termed the *internal language.*

We learn to read by associating the visual appearance of certain symbols with the sounds previously learned of respective letters and words. These visual memories are stored in the angular gyrus (Fig. 138) and vicinity. When one reads aloud the words are first recognized by the visual center, which calls up the corresponding sound in the auditory center, which in turn stimulates the motor speech center, which starts in motion the muscles whose center is in the foot of the ascending frontal convolution. When we read silently the same process takes place by means of the internal language. The patient may lose the power of recognizing letters and still recognize words, and vice versa.

In learning to write the visual perception of the letters is associated with certain coördinate muscular movements of the fingers and arm necessary to form them. These memories are kept in the *left second frontal convolution* (Fig. 138) in right-handed people, and vice versa. It is termed the *writing or cheirokinesthetic center.* In writing each letter is self-dictated by means of either the spoken or internal language; therefore the auditory and motor speech centers are first excited. Loss of the power of writing when not due to paralysis of the muscles of the arm and hand is termed *agraphia.*

All of the centers having to do with speech are connected by means of association tracts, lesions of which will cause interference with the function of the centers (Fig. 138). While a lesion may be confined to one or involve more than one center, from the above it will be seen that each center, while it has certain functions, is more or less dependent on the other. Therefore it is a fact that a lesion of one, especially if it develop suddenly, causes more or less interference with the function of the others. This has been termed by von Monakow, *diaschisis.*

The symptoms are rarely sharply limited to the functions of one center. According to whether the receptive or emissive function is affected, aphasia has been divided into *sensory* and *motor.*

A patient has sensory or *Wernicke's aphasia* if, the periphereal apparatus being intact, he is either unable to understand the language he is familiar with or to recognize the meaning of the symbols, either printed or written, with which he was once familiar. In other words, the once familiar sounds are to him as those of a foreign language, or he sees, but the symbols appear to him as those of a foreign language. The former is known also as *auditory aphasia, or word deafness,* the latter as *alexia, visual aphasia or word blindness.*

Auditory and visual aphasia are divisions of a condition known as *agnosia.* By this is meant the inability to recognize a heretofore familiar object by any of the senses. In other words, not to be able to call it by name, although the name can be pronounced when once heard. This is called *anomia.* There may be ability to do this through one or more senses and not others, or all may be lost. Thus according to the sense lost we have *auditory agnosia*, which comprises word deafness, music deafness, inability to recognize objects by any sound they may cause; *visual agnosia,* of which word blindness is one manifestation, and *tactile agnosia,* also known as astereognosis (p. 578).

Mills has placed a center which he calls the naming center in the *left second temporal convolution* in right-handed people, and vice versa. To this center tracts go from the various perceptive centers, as the auditory, visual, tactile, etc. A lesion in or near the center N (Fig. 138), will cause *complete agnosia,* if one of the tracts or centers, as SN, VN, AN will cause *visual, tactile, or auditory agnosia*, as the case may be. Agnosia is also known as *mind blindness.*

Another related condition is that known as *apraxia.* Agnosia has sometimes been termed sensory apraxia. The condition here considered is *motor apraxia, or dyspraxia.* It sometimes occurs associated with agnosia of the auditory or visual type. By it is meant the forgetting of how to perform heretofore familiar actions, motor paralysis of the limbs being absent or so slight as not to cause the disability. Thus the patient may recognize a pencil but be unable to put it to proper use. On being requested to make certain movements he may make others (*parapraxia*). In other cases he may make one movement correctly but when told to make another he repeats the first (*intentional perseveration of Liepmann*), or he may continue to make a movement or perform an action for some time after being told to stop (*clonic perseveration of Liepmann*). Various parts of the brain have caused these disturbances. Lesions of the corpus callosum have caused left-sided apraxia; it has also occurred in lesions of the left frontal lobes and left parietal lobe.

Motor Aphasia (Broca's) or aphemia is present when the patient, the peripheral speech apparatus (lips, tongue, larynx) being intact, is unable either partially or completely to give utterance to his thoughts. The lesion is in the left third frontal convolution.

Agraphia.—Loss of the power of writing may be due to a lesion of the writing center in the *second* left frontal convolution. This is termed agraphia. It may occur independently but is usually associated either with word blindness or motor aphasia.

In some cases the patient may be able to speak, but he uses wrong words, skips them—in other words, talks incoherently. This is termed *paraphasia, or conduction aphasia,* and may be due to a lesion either in the center A or the tract A-G K (Fig. 138).

Loss of the ability to make or understand gestures is known as *amimia.* There may be loss of the power in those who once possessed it to either produce or recognize musical sounds (*amusia*). This may or may not be associated with defects of ordinary speech.

The following division of aphasia has been made. As a matter of fact it is difficult to definitely localize lesions as here described, and the principle of diaschisis (p. 617) must be borne in mind. It is useful, however, in studying cases. The letters refer to Fig. 138.

CORTICAL AUDITORY APHASIA.—Lesion is at *A*. Consists of loss of power of understanding spoken words; of repeating words heard or writing them from dictation. Stimulation of this center being needed to activate *G K,* spoken speech is defective (paraphasia). For the same reason the internal language is interfered with and the power of reading and writing is defective (tracts *V-A-G K*).

SUBCORTICAL AUDITORY APHASIA.—Lesion in tract $A\text{-}A^1$, the center *A* being intact. The patient is unable to understand spoken words. Reading and writing from dictation are not well performed. Paraphasia may be present, as the patient when he pronounces a word cannot recognize whether it is done correctly or not. The internal language is intact and word memories can be recalled (*A*). Silent reading and writing are therefore not interfered with (tract *V-A-G K*).

CORTICAL VISUAL APHASIA. Lesion in center *V*. Patient has inability to read aloud or silently (alexia); to write with facility either spontaneously or from dictation and to copy understandingly. Speech is not interfered with.

SUBCORTICAL VISUAL APHASIA. Lesion in tract $V\text{-}V^1$. There is loss of ability to read and copy understandingly. Through the tract V^1 *C K* he can copy mechanically. V^1 being intact, visual memories can be recalled and he can write imperfectly as one does with the eyes closed.

CORTICAL MOTOR APHASIA. Lesion in *G K*. Spontaneous speech, repeating words and reading aloud are either partially or completely lost. Owing to the loss of the internal language, the power of silent reading and writing is also interfered with (*V-A-G K*). Language is understood.

Subcortical Motor Aphasia. Lesion in tract *G K-U*. Spontaneous speech, reading aloud and repeating words are lost. The internal language *A* and *G K* being intact, silent reading and writing are not interfered with. Language is understood.

The above is the generally accepted view of the centers controlling speech. Marie and others differ from this view. They believe the only part of the brain which controls speech to be the zone of Wernicke, which comprises the supra-marginal, angular, and posterior part of the first two temporal convolutions. A lesion in this region causes sensory aphasia, due to an intellectual deficit. They do not believe that the third frontal convolution has anything to do with speech, but that so-called *motor aphasia is sensory aphasia plus anarthria* (*infra*), the latter occurring when a lesion affects the lenticular zone, an area comprised between a line passing in a transverse direction from the anterior fissure of the island of Reil to a corresponding point in the lateral ventricle. Within this are the corpus striatum, external and internal capsules and island of Reil.

The anarthria or aphemia of Marie is characterized by loss of speech, with preservation of the understanding of words, of reading and writing. It is an interference with the coördination of movements required for phonation without motor paralysis.

Transient Aphasia may be due to functional disturbances due to severe fright, anger, migraine (p. 772), toxemiae as gout, uremia, infectious diseases, vegetable and mineral poisons, hemorrhage and exhaustion.

Diagnosis.—Before making a diagnosis of aphasia careful examination must be made to determine that sight, hearing, and the action of the muscles concerned in speech are intact.

Interference with speech due to faulty action of the muscles (lips, tongue, larynx) is known, if completely lost, as *aphemia* or *anarthria;* if partially so, as *dysarthria.* Such disturbances are seen in bulbar palsy, multiple sclerosis, cerebellar disease, paresis, sometimes during convalescence from exhausting diseases (bradylalia), in patients with cleft palate, adenoids, paralysis of the soft palate following diphtheria (p. 672), and in stammering and stuttering.

If it is believed to be a form of aphasia, the following examination should be made: (1) voluntary speech; (2) exclamatory speech; (3) ability to answer questions; (4) associative speech, as counting, repeating the alphabet, etc.; (5) quality of speech, such as confusion, incoherence, jargon; (6) repeating spoken words; (7) indicating the number of syllables in a word, to show that the patient knows it is a word; (8) writing; (9) writing to dictation; (10) copying; (11) singing or humming tunes; (12) ability to make gestures; (13) ability to understand gestures; (14) understanding spoken words, sentences and complex directions; (15) naming things seen, felt, heard, touched, smelled, tasted; (16) knowing the proper use of and manner of using objects; (17) recalling to mind objects named; (18) reading understandingly, aloud and silently; (19) reading letters and numerals; (20) reading aloud without understanding it; (21) general intelligence.

Lesions in the centrum ovale may involve motor, sensory and association fibers. If near the motor region of the cortex (subcortical lesion) motor paralysis is caused (Fig. 137). If spasms do occur, they are due to extension of the lesion (as a tumor) to the cortex and paralysis precedes their development. In cortical lesions, it is usually vice versa. If the lesion is near the internal capsule the symptoms resemble those of a lesion in that situation.

The principal symptom of localizing value in *lesions of the corpus callosum* is apraxia of the left hand and arm (p. 617). Some paralysis of hemiplegic type and pseudo-bulbar palsy (p. 708) may occur.

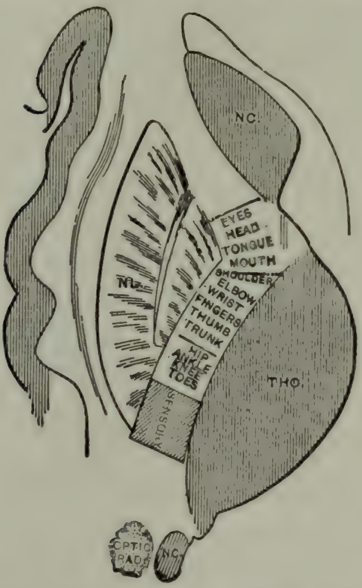


Fig. 139.—Diagram of Motor and Sensory Representation in the Internal Capsule. NL., Lenticular Nucleus; NC., Caudate Nucleus; THO., Optic Thalamus. (After Osler.)

Through the *internal capsule* pass the motor and sensory projective fibers to and from the cortex (Fig. 139). Hemiplegia is the usual symptom caused by a lesion here. Conjugate deviation of the eyes, hemianesthesia and hemianopsia due to the proximity of the optic radiations may all occur.

The *third cranial nerve* rises from the *crus cerebri* near the pyramidal tract. A lesion here causes hemiplegia of the opposite side and third nerve palsy (p. 633), of the same side (syndrome of Weber). As the optic tract crosses the crus it may be involved, causing homonymous hemianopia. If the *tegmentum* is involved hemianesthesia with or without motor paralysis is caused, and when the red nucleus is involved either tremor or athetoid movements of the affected limbs occur (Fig. 140).

There is more or less dispute concerning the functions of the so-called *basal ganglia* (*optic thalamus and corpus striatum*). Some progress is being made, however, in determining them, especially in the case of the latter. The *optic thalamus* is connected with the cortex of the frontal, motor, parietal, occipital and temporal regions; it is also connected with the corpus striatum and red nucleus. The sensory fibers from the cord probably end here, where a new neuron (the thalamic-cortico tract) begins and runs to the sensory region of the cortex; it also receives fibers from special sense paths. Together with the anterior corpora quadrigemina and the external geniculate bodies, the pulvinar forms part of the primary optic centers (p. 598). It probably also exerts an influence over various automatic functions, as emotional control and facial expression. Athetoid and choreiform movements have followed disease in it. The pain that sometimes is present in the affected limbs of a hemiplegia following apoplexy seems to be due to a thalamic lesion.

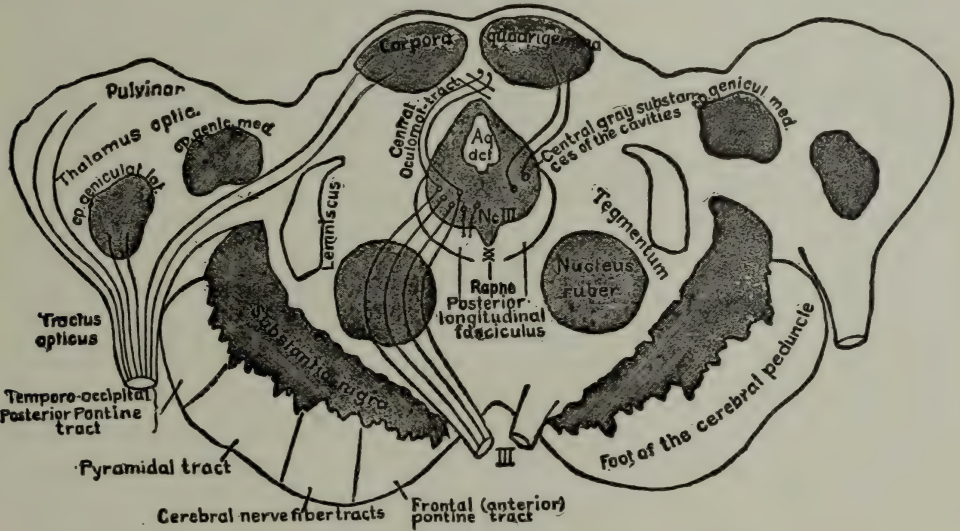


Fig. 140.—Section of the Middle Brain at the Height of the Corpora quadrigemina. (Diagram, in part, after Obersteiner.)

Thalamic lesions have caused hemianesthesia, pains in the affected side, little or no motor paralysis, absence of the Babinski reflex, hemiataxia, and choreiform movements in the affected limbs, causeless laughter and crying, paralysis of the facial muscles in expressing the emotions (as laughing) with preservation of the power of voluntary motion. Hemianopsia and hemiplegia may occur from pressure on the optic and pyramidal tracts.

The functions of the *lenticular nucleus* of the *corpus striatum* have recently been determined with some certainty (Kinnier-Wilson, Brain, 1914, 427). Its principal influence seems to be a steadying or controlling influence over muscle tone. Lesions of it exclusively, therefore, cause hypertonicity and tremor of an intention type with absence of motor paralysis (p. 688). When this is present it is due to pressure on the pyramidal tract. Causeless emotionalism may also be a symptom. Mills believes that if the *caudate nucleus* is involved the emotions are of a painful character, and in addition there are symptoms referable to the vasomotor and secretory systems with disturbances of temperature, pulse, respiration and glandular activities (Mills, Neurol., Centralbl., 1914, No. 24).

The *anterior tubercles of the corpora quadrigemina* form part of the primary optic centers (p. 598). Loss of their function may cause loss of reflex movements of the iris and ciliary body, although probably the influence of the external geniculate body is greater.

The *posterior tubercles* together with the internal geniculate bodies are connected with the auditory nerves and cerebellum. If diseased, diminution of hearing and incoördination occur. Third nerve palsy, loss of associated movements of the eyes upward, hemianopsia, and weakness of the limbs of the opposite side may be present but are due to pressure on or involvement of neighboring parts (Fig. 140).

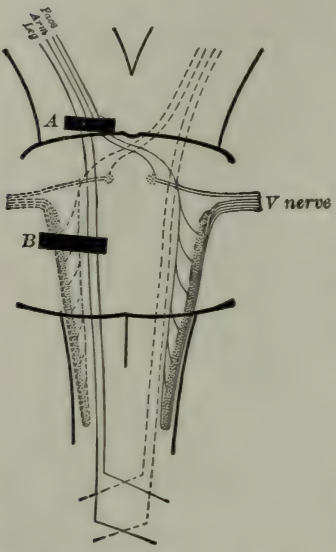


Fig. 141.—The Sensory Tract in the Crus, Pons, and Medulla, Showing Nucleus and Roots of V Nerve. A, Lesion Causes Right Hemianesthesia; B, Lesion Causes Alternating Hemianesthesia; Left Face and Right Side of Body. (After Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

The principal symptom indicating a *lesion of the pons* is *crossed* or *alternating paralysis,* i. e., cranial nerve on the side of the lesion, arm and leg on the opposite side. The cranial nerves usually affected are the sixth, seventh and eighth, although the fifth may also be affected. (Fig. 137 and Fig. 141). If the sixth is involved by an intramedullary lesion there is loss of lateral conjugate movement of the eyes to the side of the lesion (p. 636), this being a distinguishing sign from extramedullary or nerve trunk lesion in which the paralysis is confined to the external rectus.

As the pons contains sensory tracts from the cord and the descending root of the fifth nerve, hemianesthesia, which, if the lesion is below the upper third is crossed (Fig. 141), may be present.

Paralysis of the sixth and seventh nerves without the auditory indicates a lesion in the posterior part. If the middle cerebellar peduncle is involved incoördination and forced movements occur. There may be disturbances of respiratory and cardiac action and hyperpyrexia.

Lesions of the medulla, which contain the ninth, tenth, eleventh and twelfth cranial nerve nuclei and the pyramidal and sensory tracts, give origin to bulbar symptoms (p. 691), developing suddenly or gradually, as the lesion is acute or chronic. There may be motor paralysis of hemiplegic or diplegic type and if there is pressure on the floor of the fourth ventricle, polyuria, or glycosuria, or both.

The principal function of the *cerebellum* is the regulating of synergy. Lesions of it cause the various manifestations of asynergy (p. 582).

In addition to the symptoms there detailed, *asthenia* and *atonia* of the muscles frequently occur. According to Mills and Weisenburg (J. Am. Med. Ass., Nov. 21, 1914, p. 1813), the former is due to the exhaustion which results from efforts to perform movements which cannot be properly grouped and directed and the latter is a relaxation dependent on the fact that tonectic stimuli from the cerebral cortex cannot rhythmically combine with unsynergized movements. These authors believe that the maintenance of muscle tone is a cerebral function and not one of the cerebellum, as is believed by many.

There may also be speech disturbance consisting of a more or less jerky, sing-song utterance due to incoördination (asynergy) of the muscles of speech. Each lobe of the cerebellum controls the muscles of the same side. An attempt has been made to determine the particular part of the cerebellum that governs each part of the body (J. Am. Med. Ass., Nov. 21, 1914, p. 1817).

10. Spinal Localization

The spinal cord transmits motor impulses from the cortical motor centers through the pyramidal tracts to the cells in the anterior horns which give origin to the peripheral nerves, and sensory impulses from the skin, muscles and joints to the brain (See Motor and Sensory Tracts).

The cells in the anterior horns, in addition to being the beginning of the peripheral motor neuron, are reflex centers, and the trophic centers of the muscles. The posterior roots and posterior horns exercise a trophic influence over the bones, joints and skin (p. 597) (See also Tabes and Syringomyelia). It is also certain that the posterior root ganglion exercises an influence over the skin (p. 685).

The location of the centers in the cord which control vasomotor and visceral action and the connections with the sympathetic system is given

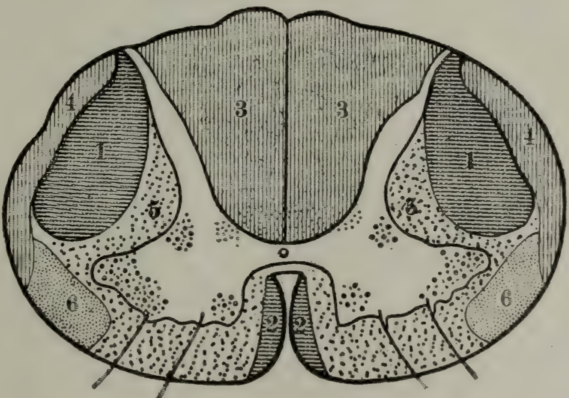


Fig. 142.—Diagram of Cross-section of the Spinal Cord. 1, Lateral Pyramidal Tract; 2, Ventral Pyramidal Tract; 3, Dorsal Columns; 4, Direct Cerebellar Tract; 5, Ventrolateral Ground Bundles; 6, Ventrolateral Ascending Tract of Gowers. (Van Gehuchten.)

on page 593. The ciliospinal center is located in the eighth cervical and first dorsal segments (pupillary skin reflex) (p. 589).

In the lower part of the cord (conus medullaris) are located the spinal automatic centers, i. e., the genital center presiding over the functions of erection and ejaculation, and the bladder and the rectal centers, which control the functions of urination and defecation (pp. 592, 629).

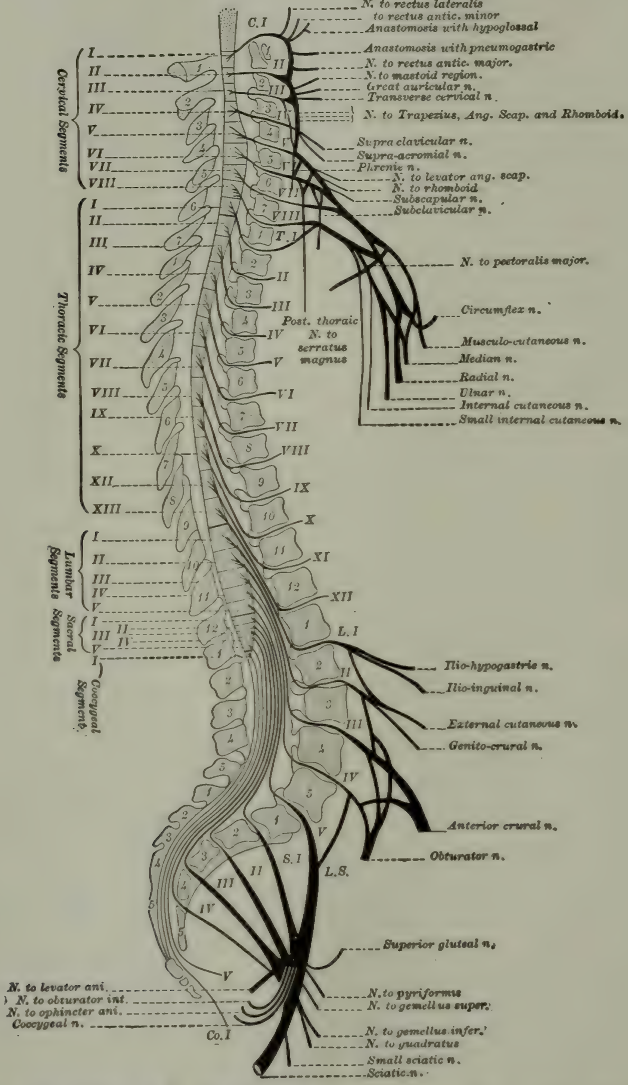


Fig. 143.—The Relations of the Segments of the Spinal Cord and Their Nerve Roots to the Bodies and Spines of the Vertebrae. (After Starr; from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

The spinal cord, therefore, may be considered a series of thirty-one divisions known as *segments* placed one above the other, each of which is connected with a pair of spinal nerves, and contains trophic, vasomotor, secretory and reflex centers and through which pass the motor and sensory tracts crossing from and to the brain respectively. Impulses inhibiting and regulating the reflexes also pass from the brain through the cord (Fig. 142).

It must be remembered that the functions of these segments overlap and that destruction of one segment will not cause symptoms referable to that segment unless the one above and one below are also damaged. A focal lesion will not damage a single muscle but will involve several.

As the spinal cord reaches from the foramen magnum to the base of the first lumbar vertebra, it is apparent that each segment is not opposite the vertebra whose name it bears, and that the intraspinal course of the nerves, after they emerge from the cord, is longer the lower we descend (Fig. 143). Hence the nerve roots from the lumbar and sacral segments form a large bundle (cauda equina), each root of which runs from its origin through the spinal canal to the vertebrae between which it emerges.

Chipault has given the following rules for the determination of the relation of the segments to the spinous processes of the vertebra:

> "In the cervical region add one to the number of the vertebra and this will give the number of the segment opposite to it; in the upper dorsal region add two; from the sixth to the eleventh dorsal add three. The lower part of the eleventh dorsal spinous process and the space below it are opposite the lower three lumbar segments. The twelfth dorsal spinous process and the space below it are opposite the sacral segments" (Fig. 143).

The most important parts of the cord are the cervical and lumbar enlargements, which give origin to the nerves supplying the muscles of the arms and legs respectively and contain the centers for important reflexes. The former comprises the segments from the fourth cervical to the first dorsal inclusively, the latter from the first lumbar to the fifth sacral. By knowing the particular functions of each segment we are able to locate the situation and extent of a focal lesion in the cord.

The following table from Osler's "Practice of Medicine," made originally by Starr and elaborated by others, shows our present knowledge of the *functions* of each segment:

LOCALIZATION OF THE FUNCTIONS IN THE SEGMENTS OF THE SPINAL CORD

Segment	Striped Muscles	Reflex	Skin-Fields (See Plate VII)
I, II and III C	Splenius capitis Hyoid muscles Sternomastoid Trapezius Diaphragm (C III–V) Levator scapulæ (C III–V)	Hypochondrum (?) Sudden inspiration produced by sudden pressure beneath the lower border of ribs (diaphragmatic)	Back of head to vertex Neck (upper part)

Localization of the Functions in the Segments of the Spinal Cord—*Continued*

Segment	Striped Muscles	Reflex	Skin-Fields (See Plate VII)
IV C	Trapezius Diaphragm Levator scapulae Scaleni (C IV–T I) Teres minor Supraspinatus Rhomboid	Dilatation of the pupil produced by irritation of neck. Reflex through the sympathetic (C IV–T I)	Neck (lower part to second rib) Upper shoulder
V C	Diaphragm Teres minor Supra and infra spinatus (C V–VI) Rhomboid Subscapularis Deltoid Biceps Brachialis anticus Supinator longus (C V–VII) Supinator brevis (C V–VII) Pectoralis (clavicular part) Serratus magnus	Scapular (C V–T I) Irritation of skin over the scapula produces contraction of the scapular muscles Supinator longus and biceps Tapping their tendons produces flexion of forearm	Outer side of shoulder and upper arm over deltoid region
VI C	Teres minor and major Infraspinatus Deltoid Biceps Brachialis anticus Supinator longus Supinator brevis Pectoralis (clavicular part) Serratus magnus (C V–VIII) Coracobrachialis Pronator teres Triceps (outer and long heads) Extensors of wrist (C VI–VIII)	Triceps. Tapping elbow tendon produces extension of forearm Posterior wrist. Tapping tendons causes extension of hand (C VI–VII)	Outer side of forearm, front and back Outer half of hand (?)
VII C	Teres major Subscapularis Deltoid (posterior part) Pectroalis major (costal part) Pectoralis minor Serratus magnus Pronators of wrist Triceps Extensors of wrist and fingers Flexors of wrist Latissimus dorsi (C VI–VIII)	Scapulohumeral. Tapping the inner lower edge of scapula causes abduction of the arm Anterior wrist. Tapping anterior tendons causes flexion of wrist (C VII–VIII)	Inner side and back of arm and forearm Radial half of the hand
VIII C	Pectoralis major (costal part) Pronator quadratus Flexors of wrist and fingers Latissimus Radial lumbricales and interossei	Palmar. Strokimg palm causes closure of fingers	Forearm and hand, inner half
I T	Lumbricales and interossei Thenar and hypothenar eminences (C VII–T I)		Upper arm, inner half
II to XII T	Muscles of back and abdomen Erectores spinæ (T I–L V) Intercostals (T I–T XII) Rectus abdominis (T V–T XII) External oblique (T V–XII) Internal oblique (T VII–L I) Transversalis (T VII–L I)	Epigastric. Tickling mammary region causes retraction of epigastrium (T IV–VII) Abdominal. Stroking side of abdomen causes retraction of belly (T IX–XII)	Skin of chest and abdomen in oblique dorsoventral zones. The nipple lies between the zone of T IV and T V. The umbilicus lies in the field of T X

Localization of the Functions in the Segments of the Spinal Cord—*Continued*

Segment	Striped Muscles	Reflex	Skin Fields (See Plate VII)
I L	Lower part of external and internal oblique and transversalis Quadratus lumborum (L I–II) Cremaster Psoas major and minor (?)	Cremasteric. Stroking inner thigh causes retraction of scrotum (L I–II)	Testicle, skin over lowest abdominal zone, groin and front of scrotum
II L	Psoas major and minor Iliacus Pectineus Sartorius (lower part) Flexors of knee (Remak) Abductor longus and brevis		Front of thigh
III L	Sartorius (lower part) Abductors of thigh Quadriceps femoris (L II-L IV) Inner rotators of thigh Abductors of thigh	Patellar tendon. Tapping tendon causes extension of leg. "Knee-jerk"	Front and inner side of thigh
IV L	Flexors of knee (Ferrier) Quadriceps femoris Adductors of thigh Abductors of thigh Extensors of ankle (tibialis anticus) Glutei (medius and minor)	Gluteal. Stroking buttock causes dimpling in fold of buttock (L IV–V)	Mainly inner side of thigh and leg to ankle
V L	Flexors of knee (ham-string muscles) (L IV–S II) Outward rotators of thigh Glutei Flexors of ankle (gastrocnemius and soleus) (L IV–S II) Extensors of toes (L IV–S I) Peronæi		Back of leg, and part of foot
I to II S	Flexors of ankle (L V–S II) Long flexor of toes (L V–SII) Peronæi Intrinsic muscle of foot	Foot reflex. Extension of Achilles tendon causes flexion of ankle (S I–II). Ankle-clonus Plantar. Tickling sole of foot causes flexion of toes or extension of great toe and flexion of others	Back of thigh, leg, and foot; outer side
III to V S	Perineal muscles Levator and spincter ani (S I–III)	Vesical and anal reflexes	Skin over sacrum and buttock Anus Perinæum. Genitals Except testicle and skin of anterior part of the scrotum

The segmental areas of sensation are shown in Plate VII.

A true *segmental type of sensory loss* only occurs when the lesion is extramedullary and involves the posterior roots. If the lesion is transverse, i. e., involves an entire section of the cord, sensory impulses cannot get through the diseased area from below, and hence the sensory loss is found not only in the area supplied by the segments involved but in that supplied by all the segments below it. For instance, if the ninth dorsal segment was destroyed, the area of skin supplied by that segment would be involved and also that supplied by all the segments below, which in this case would mean both legs and the trunk to a line passing through the umbilicus. Anesthesia due to peripheral nerve lesions is confined to the distribution of these nerves in the skin, and differs in distribution from that due to segment lesion (Fig. 123) (See also Figs. 154-157).

Often at the level of the lesion there is a zone of hyperesthesia due to irritation of the posterior roots by the lesion.

Sensory loss extending to the umbilicus corresponds to the *ninth dorsal* segment; one inch below this to the *tenth.* In lesions at this segment when the patient attempts to raise the shoulders from the bed, the umbilicus will be noticed to move upwards a half inch or more. This is due to the fact that a lesion located here causes paralysis of the recti muscles below the umbilicus only; therefore the effort to raise the shoulders causes contraction of the recti above the umbilicus and it is pulled upward.

The nipples correspond to the *fourth dorsal.* Loss of sensibility in the arms is not present if the lesion is below the *first dorsal.*

A lesion that damages the *fifth cervical segment* and spares those below causes a characteristic symptom group. The fifth segment supplies principally the biceps, brachialis anticus, deltoid and supinators. If these muscles, therefore, are paralyzed the elbow will be next to the body, and the forearm and hand will be prone on the chest. The biceps jerk will be lost, but the triceps jerk will be preserved (Fig. 144). If, however, the *fifth segment* escapes and the lesion extends to and involves the *sixth cervical segment,* the elbow will be abducted by the deltoid, the forearm flexed by the biceps, the supinators will supinate the forearm and the infraspinatus will rotate the humerus externally. The biceps jerk will be present and the triceps jerk lost. The position is seen in Figure 145.

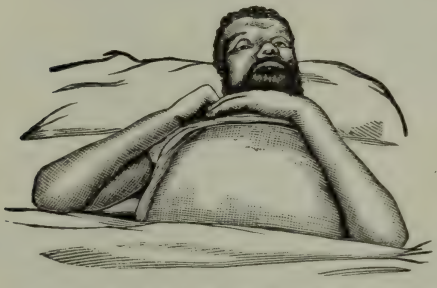
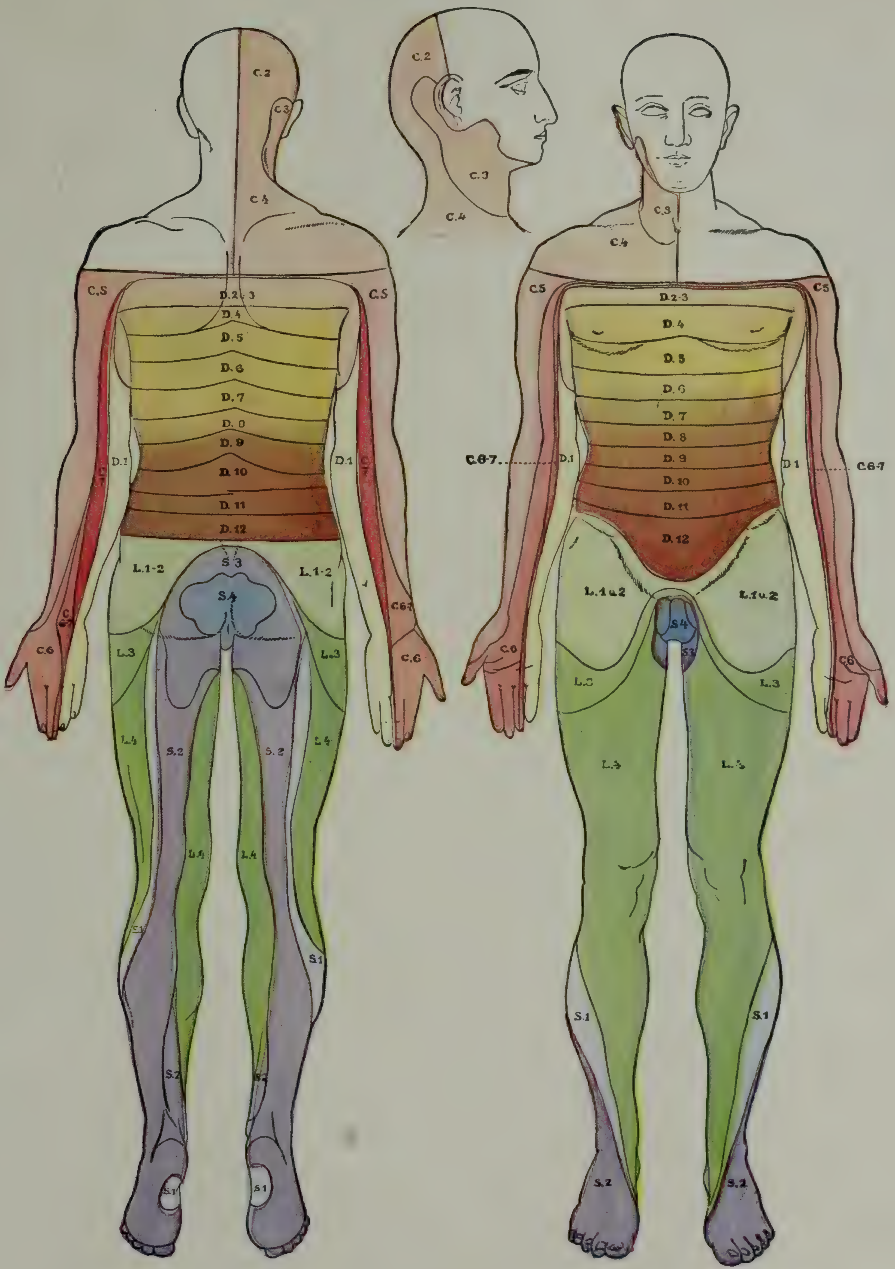


Fig. 144.—An Attitude in a Case in Which the Fifth Cervical Nerve Had Been Injured on the Left Side Only. (After Thorburn.)

That part of the cord between the fourth and fifth lumbar as the upper, and the second and third sacral segments on the lower boundary is known as the *epiconus medullaris.* A lesion here causes the following symptoms: knee jerks present; Achilles jerks absent; paralysis principally of the peroneal muscles, which are atrophied and show D R, causing a steppage gait (p. 672) and sensory loss in the area supplied by these segments and possibly the segments below, depending whether the lesion is intra- or extra-medullary.

That part of the cord between and including the third sacral segment and the filum terminale is known as the *conus medullaris.* A lesion here

PLATE VII



Distribution of the Areas of the Sensory Roots upon the Surface of the Body. (From Kocher.)

Red: Area of the cervical roots (C. 2 to C. 7).
Yellow: Area of the dorsal roots (D. 1 to D. 12).
Green: Area of the lumbar roots (L. 1 to L. 4).
Blue: Area of the sacral roots (S. 1 to S. 4).

causes paralysis of the bladder and rectal sphincters (p. 592), causing incontinence, loss of sexual power, a saddle-shaped area of sensory loss involving the skin about the arms, perineum, posterior part of scrotum, skin of penis, mucous membrane of urethra and arms. The testicle is sensitive, also the skin of the front part of the scrotum, their nerve supply being from the first lumbar.

Lesions of the *cauda equina* may simulate lesions of the *epiconus* and *conus.* The distinction is important, although sometimes difficult, as cauda lesions are more amenable to surgical interference than are those of the cord; therefore the prognosis is better.

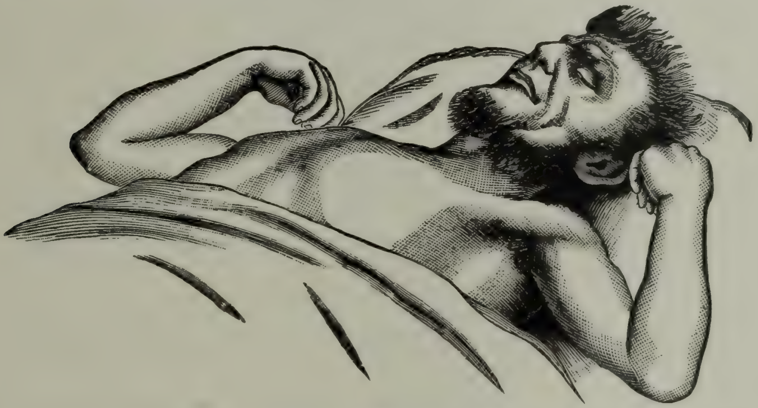


Fig. 145.—Peculiar Attitude of a Patient in Whom the Fifth Cervical Nerves Had Not Been Crushed. (After Thorburn.)

The following *differential points* are modified from Williamson:

1. If the symptoms are not due to trauma, a sudden onset or a sudden extension of the symptoms is in favor of lesion of the conus; a gradual onset in favor of lesion of the cauda equina.

2. In traumatic cases, if the seat of the injury be the upper lumbar region of the spine or if the spinous processes of the twelfth dorsal or first and second lumbar vertebrae be depressed or displaced, probably the lesion is in the conus. If the injury be in the inferior lumbar region or adjacent part of the sacrum, the lesion is probably in the cauda equina.

3. Pain at the level of the twelfth dorsal and first and second lumbar spines, which is produced or increased by pressure, percussion, or by the application of heat or cold, and pain which does not radiate into the legs, point to lesions of the cord, while pain below the level just mentioned, which is increased by pressure over the sacrum and by movement and pain radiating into the legs point to lesion of the cauda equina.

4. Very severe pain in the distribution of the nerves of the sacral plexus (sacrum, bladder, perineum, anus, and parts supplied by the sciatic nerves) is in favor of lesion of the cauda equina; absence of pain in favor of lesion of the conus. Moderate pain may occur in either.

5. Pain which precedes other symptoms for a long period is in favor of lesion of the cauda equina.

6. Anesthesia symmetrical in distribution, early and intense muscular atrophy, with reaction of degeneration (involvement of gray matter), and early onset of bedsores, are in favor of lesion of the lower part of the cord.

7. In favor of lesion of the cauda equina are asymmetrical distribution of anesthesia, progressive onset of bladder and rectal trouble, the alternation of increase and decrease in the severity of the chief symptoms, slow onset and diffuse character of the motor paralysis and muscular atrophy, and absence of bedsores.

Marked anesthesia is in favor of conus lesion. Pain is the most important symptom and usually precedes for a considerable time the development of motor symptoms. Early paralysis of the sphincter is frequent in lesions of the cauda, but may be absent. Loss of muscular power frequently shows itself first in the peroneal and tibialis anticus muscles.

Disease of either of the sacrum or sciatic nerves may be confounded with disease affecting the cauda equina, such being often called sciatica for a long time before the true trouble is recognized. For the diagnostic points see page 665.

As an example of the application of these principles we will suppose a case in which there is complete paralysis of both legs with atrophy of the flexors and abductors of the thigh and extensors of the leg on the thigh and the presence of D R. The muscles below the knee are not atrophied. Preservation of the abdominal reflex, loss of the cremasteric and patellar reflexes, preservation of the plantar reflex, which gives the extensor response (Babinski's reflex), and the presence of ankle clonus, incontinence of urine and feces, the patient being ignorant of when either the bladder or rectum is full, and sensory loss, pain, tactile temperature involving both legs to a line extending around the body on a line with the groins.

Such symptoms would point to a transverse lesion between the first and third lumbar vertebra, because reference to the table (p. 627) will show that the abdominal reflex being present the twelfth dorsal segment and above are intact; the loss of the cremaster and patellar reflexes shows that the first to third lumbar segments are diseased; the presence of ankle clonus shows the fifth lumbar segment to be intact.

The muscles atrophied are those whose nerve supply originates in the first to third lumbar segments inclusive. They are atrophied and show the D R because the lesion extending across the cord involves the gray matter; hence the paralysis of these muscles is due to a peripheral neuron lesion (p. 563).

The paralysis of the muscles below the knee is due to cutting off the pyramidal tracts above the location of the cells in the gray matter which give origin to the nerves supplying them; in other words, a lesion of the upper motor neuron; hence motor impulses cannot reach them. For the same reason ankle clonus is present because inhibition is cut off by the lesion.

Lesions that irritate the nerve roots cause a zone of *hyperesthesia* in the skin area supplied by them. This may be felt as the sensation of a tight band about the body.

In *complete transverse lesions,* i. e., those in which the cord is completely divided, the *deep reflexes* below the seat of the lesion *are lost* and the paralysis is flaccid without the existence of atrophy. The Babinski reflex may be present.

Hemilesions of the cord cause a characteristic symptom group known as *Brown-Séquard paralysis.* This is well shown in the following table modified from Gowers:

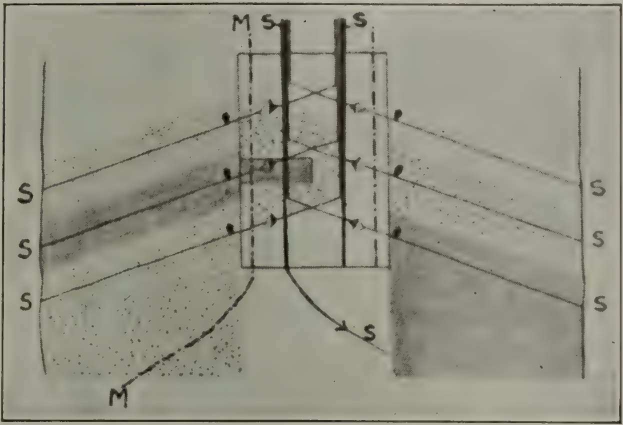


Fig. 146.—Diagram to Indicate Symptoms in a Unilateral Lesion of the Spinal Cord. Shaded Area = Anesthesia; Dotted Area = Hyperesthesia; M = Path for Motor Fibers and Muscular Sense; S = Peripheral Sensory Nerves and Sensory Tracts in Cord. The Unilateral Lesion Is Indicated in the Left Half of the Cord. The Dotted Area Around the Lesion Indicates Irritation of Nerve Fibers. The Band of Anesthesia on the Side of the Lesion Is Caused by Destruction of the Peripheral Nerve S, as It Passes Into the Lesion; the Anesthesia on the Opposite Side by Destruction of the Sensory Tracts, Containing the Fibers which have Decussated; the Hyperesthesia by Irritation of Sensory Nerve Fibers and of the Sensory Tracts Around the Lesion (Indicated by the Dotted Area. (After Williamson's "Diseases of Spinal Cord," published by the Oxford Press, London.)

	Cord	
Zone of cutaneous hyperesthesia Zone of cutaneous anesthesia Lower segment type of paralysis with atrophy	Lesion	
Upper segment type of paralysis Hyperesthesia of skin Muscular sense and allied sensations impaired Reflex action first lessened and then increased Surface temperature raised		Muscular power normal Loss of sensibility of skin to pain and temperature Muscular sense normal Reflex action normal Temperature same as that above lesion

Reference to Figure 146 will illustrate why the symptoms occur. For a short time there may be diminished tactile sensibility on the side of the lesion. These symptoms may vary some, as a lesion is not apt to be absolutely limited to one-half of the cord. They may be caused by tumors, syphilis of the cord, gunshot and stab wounds, hematomyelia, and rarely in syringomyelia.

D. Diseases of Peripheral Nerves

The important pathological conditions of the nerves are inflammation or neuritis, compression, traumatic division, and degeneration.

Before discussing these a brief description of the *symptoms caused by a lesion of each nerve* is essential.

1. Cranial Nerves

(*a*) *Olfactory Nerves*

Their functions may be disturbed by a lesion anywhere in their course, from the nasal mucous membrane to the centers in the uncinate gyrus (Fig. 136). The disturbance may be manifest by subjective sensations of smell (hallucinations) called *parosmia,* increased sensitiveness or *hyperosmia,* and loss of the sense or *anosmia.* Loss of taste is frequently associated with the latter.

Parosmia is found in the insane, in epileptics in whom the aura may be so represented, and in disease, as tumor of the uncinate gyrus and vicinity. Rarely after head injuries the sense may be perverted.

Hyperosmia usually occurs in nervous, hysterical individuals.

Anosmia may be caused by catarrhal affections of the nasal mucous membrane; lesions of the bulb or tract which may be due to falls or blows on the head, caries of the cranial bones, meningitis or tumor; lesions, usually tumor, of the olfactory centers (uncinate gyrus), and in congenital cases failure of their development.

(*b*) *Ocular Nerves*

i. Optic Nerve

This has been discussed on page 597, et sequentur.

ii. Motor Nerves of the Eyeball

These are the third or motor oculi; the fourth or pathetic, and sixth or abducens. They are pure motor nerves and may be diseased separately or collectively and the seat of the lesion may be in either the nuclei or nerve trunks.

The cortical centers for these nerves are probably in the second frontal convolution, the fibers from which pass through the internal capsule, the fibers of the third and sixth decussating before reaching the nuclei (See Fourth Nerve). Methods of examination will be found on page 601.

(1) *Third or Motor Oculi*

This nerve arises from a nucleus consisting of a number of distinct nests of cells, each of which gives origin to nerve fibers supplying a definite muscle.

The arrangement of these nests and the muscles they supply are from before backwards: sphincter iridis, ciliary muscle, convergence center, rectus superior, rectus internus, levator palpebræ superioris, obliquus inferior, and rectus inferior.

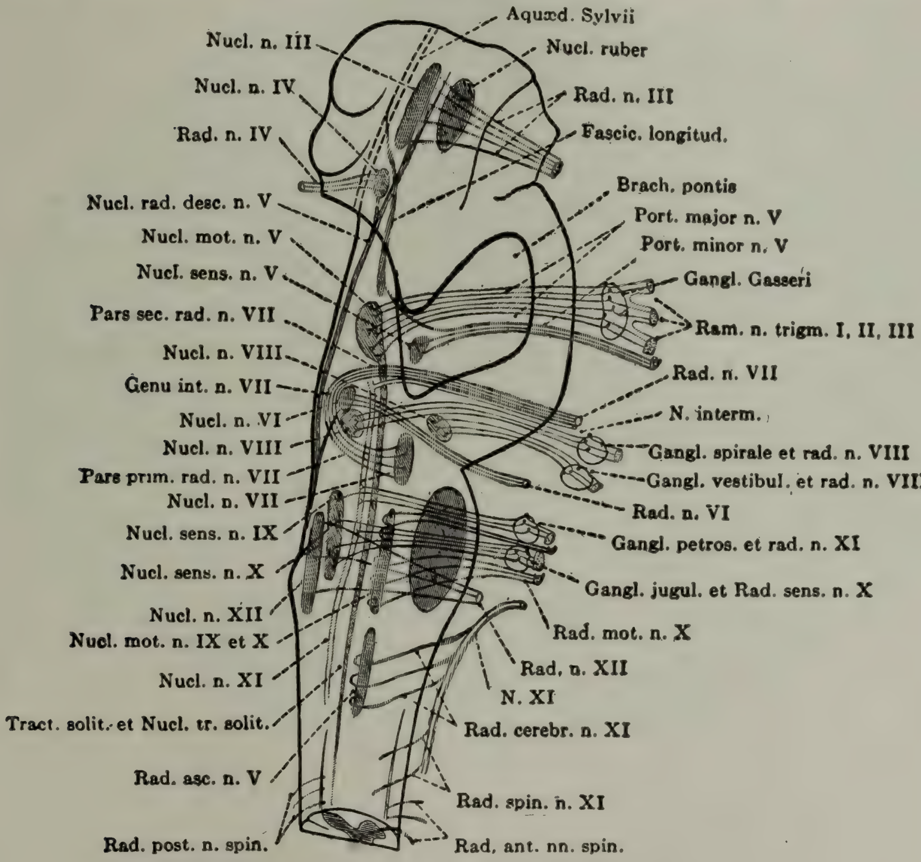


Fig. 147.—Nuclear Origin of the Cerebral Nerves. (After Edinger.)

It will be seen, therefore, that the nerve supplies all the muscles of the eyeball excepting the superior oblique (fourth nerve) and external rectus (sixth nerve).

The nuclei are situated along the floor of the aqueduct of Sylvius,

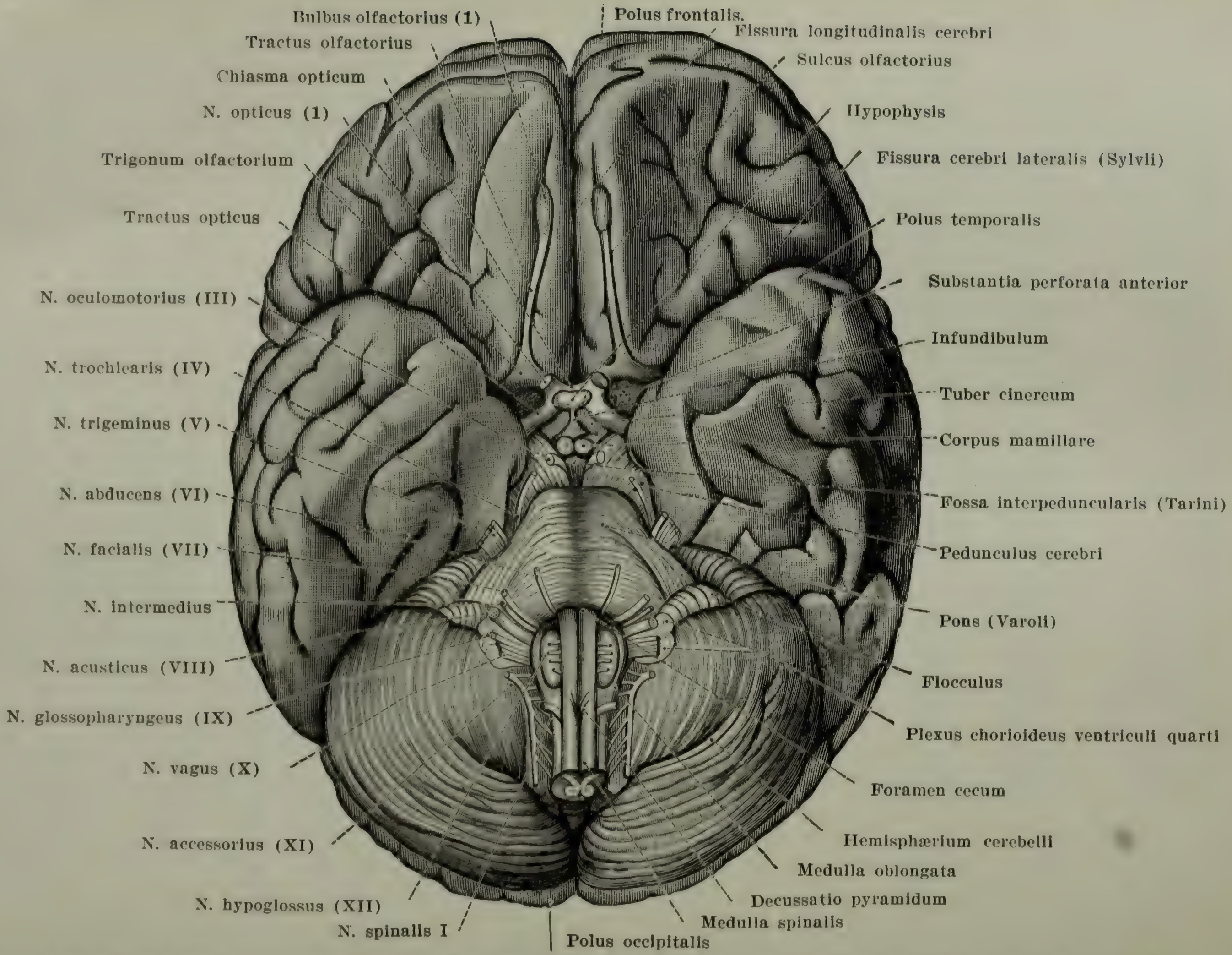


Fig. 148.—The Base of the Brain, Showing the Origin of the Roots of the Cranial Nerves. The Basal Surface of the Cerebrum in Its Posterior Division Is Covered by the Cerebellum.

beneath the corpora quadrigemina (Fig. 147). From here the nerve passes through the crus, which is its peripheral origin (Fig. 148). The nerve then passes through the cavernous sinus and enters the orbit by the sphenoidal fissure, where it divides into a number of branches to supply the muscles above mentioned.

Paralysis of this nerve if complete is known by the following symptoms: drooping of the upper eyelid (ptosis,[1]) deviation of the eyeball outward and downward (action of the external rectus and superior oblique), dilatation of the pupil, which does not contract when stimulated by light or convergence. Owing to the weakness of most of the muscles which keep it in place there may be slight exophthalmus.

The patient will complain of diplopia (p. 601).

The paralysis may not be complete; it may be limited to the extraocular muscles, some of which may escape (ophthalmoplegia externa), or the iris and ciliary muscle may be alone affected (ophthalmoplegia interna).

Paralysis of the iris (pupillary rigidity) is known as *iridoplegia.* If limited to lack of response to light only it is *reflex iridoplegia;* if to convergence and accommodation only, *cycloplegia.* Reflex iridoplegia is also known as Argyll-Robertson pupil (p. 592). Methods of examination will be found on pages 565 and 601.

(2) *Fourth or Pathetic Nerve*

The nuclear origin is just below the third (Fig. 147). The fibers differ from the other cranial nerves in decussating as soon as they leave the nucleus. The nerve supplies the superior oblique muscle.

Isolated paralysis of this nerve is rare; when present the eye is rotated upwards and inwards. Diplopia of the homonymous variety is present (p. 601).

(3) *Sixth or Abducens Nerve*

This arises from a nucleus in the floor of the fourth ventricle. It is practically surrounded by the fibers of the seventh nerve (Fig. 147). Its superficial origin is between the pons and medulla (Fig. 148). It passes through the cavernous sinus and enters the orbit through the sphenoidal fissure to supply the external rectus muscle.

Through the posterior longitudinal fasciculus the sixth nerve nucleus is connected with the division of the third, supplying the internal rectus on the opposite side (p. 636).

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[1] Ptosis is rarely caused by disease of the sympathetic, causing paralysis of the smooth muscle fibers in the fascia of the orbit; the symptoms detailed on page 594 would also be present. Apparent ptosis may be due to spasm of the orbicularis muscle (p. 644).

Through this also the muscles of the eye are connected with Deiters' nucleus and the vestibular division of the eighth and the cerebellum, this accounting for vertigo sometimes occurring when there is weakness of the ocular muscles (p. 646) and nystagmus occurring in cerebellar disease (p. 586).

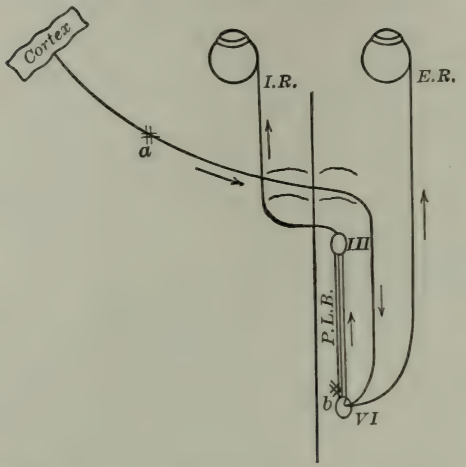

Fig. 149.—Diagram Showing the Probable Relations of the Nuclei of the Sixth and of the Internal Rectus Branch of the Third to the Brain. P.L.B., Posterior Longitudinal Bundle. (After Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

Paralysis of this nerve is known by the presence of convergent squint and homonymous diplopia (p. 601). If the lesion (*b* in Fig. 149) causing the paralysis is either in the nucleus or posterior longitudinal fasciculus (lesion in the pons) there will be weakness of the internal rectus of the opposite eye, and hence loss of conjugate lateral movement of the eyes to the side of the lesion. The eyes look *away from* the lesion. If, however, the lesion is above the nucleus (*a* in Fig. 149) or in the cortex, there is inability to move both eyes laterally away from the side of the lesion. The eyes look *toward* the lesion. The loss of conjugate lateral movement indicates a lesion either in the pons or above the nucleus, therefore paralysis of the external rectus without this symptom is due to a lesion of the nerve trunk.

Etiology.—Paralysis of any of these nerves may be caused by neuritis due to any of the causes of multiple neuritis (p. 670), but especially diphtheria; traumatism causing fracture at the base through the anterior fossa or sphenoidal fissure, or hemorrhage; pressure on the nerve by the exudation of meningitis, usually syphilitic; pressure of a tumor in the region of the nuclei (pp. 620, 622); at the base of the brain (Fig. 148) or within the orbit;[1] degeneration due to arteriosclerosis, tabes dorsalis, paresis and multiple sclerosis;[2] inflammation of the nuclei (polioencephalitis superior of Wernicke) due to infectious febrile diseases, especially influenza, or associated with acute poliomyelitis; ptomain poisoning, lead, alcohol, Basedow's disease and syphilis; hemorrhage from or thrombosis or embolism in the arteries supplying the nuclei (branches of posterior cerebral or basilar); chronic nuclear degeneration associated with bulbar palsy (p. 691) or progressive spinal muscular atrophy; *thrombosis of the cavernous sinus,* where it will be associated with exophthalmus, dilatation of the frontal veins, cyanosis and edema of the orbital and frontal regions and involvement of the ophthalmic division of the fifth cranial nerve; sometimes in cerebellar disease, when it is manifested as "skew deviation," i. e., one eye is directed downwards and the other upwards; congenital, usually manifested as ptosis. Apparent paralysis due to hysteria is certainly in most instances due to spasm (p. 638). Paralysis from this cause is said to occur by some, but if it does, is exceedingly rare.

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[1] Paralysis of the external rectus may be due to tumor anywhere in the brain. This is due to the fact that the course of the sixth nerve is so long that anything which distorts the brain may cause pressure or pulling. It is often involved in tumors of the cerebellopontile angle (p. 722).

[2] This form may be transient and due to arterial spasm (p. 673). Diplopia may be one of the earliest symptoms of tabes and multiple sclerosis.

Transitory paralysis of these nerves has occurred after the induction of spinal anesthesia. There is also a *recurrent form* which may eventually become chronic and is associated with migraine (p. 772). *A rare progressive family disease* in which there is ptosis and paralysis of the glossopharyngeal and vagus nerves has been described (Taylor, Journal of Nervous and Mental Diseases, March, 1915, p. 129).

Paralysis of any or all of these nerves of acute onset is usually due either to hemorrhage, embolus, thrombosis or inflammation (*supra*). When due to the latter there would be headache, vertigo, vomiting, pos-

Nuclear	Nerve
Isolated paralysis of individual or a few muscles Progressive development of symptoms Absence or slight development of ptosis	Usually all muscles supplied by the particular nerve involved are paralyzed at the same time. Rarely this is not so, especially in orbital or basal lesion. In the latter situation the nerve is in a number of small branches before becomimg one trunk
Symptoms usually bilateral	Frequently unilateral; may, however, be either
Association of paralysis with other degenerative cerebral or spinal disease	Not associated with other cerebral or spinal disease, except disease at the base of the brain, when other cranial nerves in the neighborhood may be involved
Escape of intra-ocular muscles	Intra-ocular muscles rarely not affected
If sixth nerve is involved, coexistence of loss of conjugate lateral movement of the eyes (this may also be due to lesion above the nucleus [p. 636])	Unless the nerves of both sides are involved, loss of associated movements does not occur
Paralysis of associated upward movement of the eyes (p. 721)	
Sudden or instantaneous onset due to hemorrhage or thrombosis	Unless due to traumatism, the onset is not instantaneous, as in apoplexy; it may, however, be acute

sibly fever, followed by stupor, possibly coma, and rapid development of the paralysis.

The most *common cause of paralysis of the ocular muscles* is probably syphilis. It also follows diphtheria with some frequency but is rarely complete, ophthalmoplegia interna being usually found.

Chronic Disease of the Nuclei (*Ophthalmoplegia*).—This may follow the acute, but is generally a degeneration existing independently or in association with progressive spinal muscular atrophy and bulbar palsy. It may also be due to syphilis, infective fevers, diabetes, and is sometimes congenital.

Symptoms.—The symptoms consist of a gradual and progressive involvement of either several or all of the ocular muscles. Ptosis is frequently absent or incomplete. It is usually bilateral, but may be unilateral.

Whether the seat of the lesion is in the nerve trunks or in the nuclei is often difficult to determine. The table on page 637 shows the chief symptoms used in distinguishing between the two, but there may be exceptions.

iii. Spasm of the Ocular Muscles

These may be either tonic or clonic, and functional or organic in origin.

The causes of *functional spasm* are hysteria, neurasthenia and errors in refraction. Strabismus may be so caused in hysteria and pseudoptosis may be due to tonic spasm of the orbicularis palpebrarum muscle (see p. 644). It can usually be told by the resistance to raising the upper lid and by the existence of other symptoms of the conditions mentioned.

When of organic origin, it is due to irritation somewhere in the tracts from the cortical centers to nerve endings in the muscles. Conjugate deviation of the eyes is the usual manifestation and may be due to irritative lesions in the same locations that cause paralytic deviation; the eyes of course will then be directed in the opposite direction (p. 636). It is most frequently seen just after an apoplectic attack.

Apparent spasm may be one of the manifestations of tic (p. 764).

(*c*) *Facial Nerves*

1. *Fifth or Trifacial Nerve*

This nerve consists of two parts—motor and sensory. The cortical center of the motor portion is in the lower portion of the precentral convolution, near those for the lips and tongue. From here the fibers pass downward through the knee of the internal capsule and after decussating in the medulla join the motor nucleus there. From this the motor root arises which becomes part of the inferior maxillary division and supplies the muscles of mastication—masseter, temporal, pterygoids; also the mylohyoid, anterior portion of the digastric and tensor tympani.

The sensory portion arises from the cells of the Gasserian ganglion, this being analogous to the posterior root ganglia (p. 556). The afferent portion enters the pons and divides, one division (the ascending root) passing up to nearly the third nerve nucleus, the other passing down (descending root) into the cord to about the second cervical segment. The fibers of both roots end in the substantia gelatinosa and from these fibers enter the fillet, decussate and ascend to the brain cortex (ascending parietal convolution). Collaterals from the descending root go to the nuclei of the motor cranial nerves.

The efferent portion divides into three parts: the ophthalmic, superior maxillary, and inferior maxillary nerves.

The sensory divisions supply the skin of the face and head to about the coronal suture (Fig. 150), most of the dura (p. 571), the conjunctiva and mucous membrane of the mouth, tongue, nose, upper part of the pharynx and teeth. Secretory fibers probably go to the lacrimal and salivary glands. Whether this nerve conducts sensation of taste or not is a disputed point (See p. 648).

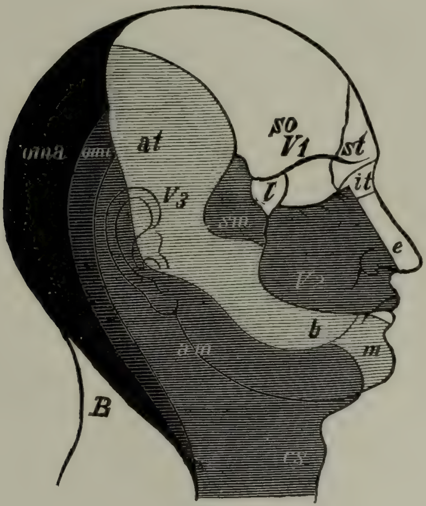

Fig. 150.

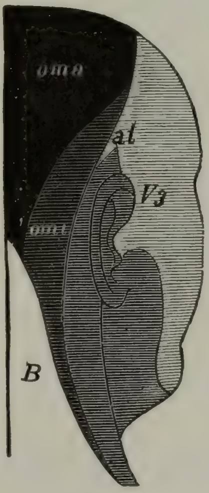

Fig. 151.

Figs. 150 and 151.—Distribution of the Sensory Cutaneous Nerves in the Head. *oma* and *omi*, Occipitalis Major and Minor; *am*, Auricular magnus; *cs*, Superficial Cervical; V_1, V_2, V_3, First, Second, and Third Branches of the Fifth (V); *so*, Supraorbital; *st*, Supratrochlear; *it*, Infratrochlear; *e*, Ethmoidal; *l*, Lachrimal; *sm*, Subcutaneous malae, or zygomatic; *at*, Auriculo-temporal; *b*, Buccinator; *m*, Mental; *B*, Posterior Branches of the Third Cervical. (After Strümpell.)

The different areas supplied by each division are shown in those marked V_1, V_2 and V_3 in Figure 150.

Paralysis may involve the entire nerve or either the motor or sensory division, or one of the divisions of this may be affected. The *entire nerve* may be involved by lesions at the base, as tumor in the cerebellopontile angle, meningitis due to syphilis or other cause. A complete paralysis of the *sensory portion* only may be due to one of these causes or to disease of the Gasserian ganglion.

The *ophthalmic division* lies in the cavernous sinus and may be affected by thrombosis there (p. 637) or by lesions at the sphenoidal fissure. Tumors of the pituitary region may also affect it. Within the orbit either new growths or inflammatory processes may injure it.

The *superior and inferior maxillary* divisions lie in the sphenomaxillary fossa and may be injured by fracture and tumors in this region and from tumors in the middle cranial fossa.

Either tumor, hemorrhage, softening (thrombosis posterior inferior cerebellar artery) or a patch of sclerosis (in multiple sclerosis) in the pons may damage the descending root. If the lesion is here there is crossed paralysis (p. 622) and other nerves, especially the sixth (p. 635), may be involved.

Traumatism to the nose or mouth may injure some branches.

Neuritis due to exposure, rheumatism, gout, and syphilis may be a cause in rare cases.

Symptoms.—The symptoms of paralysis of the sensory division is loss of sensation in all parts supplied by it, or if only one division is affected, the parts supplied by it.

Pain sense is usually lost first. According to Spiller, there are only pain and temperature fibers in the descending root and there is a separate tract here for each of these, so that one may be involved without the other. *Pressure sense* is not lost (pp. 578 and 641).

Neuralgic pain may precede the development of sensory paralysis (See Neuralgia), and such pain plus loss of sensation is significant of a destructive lesion affecting the nerve at the base of the brain or the Gasserian ganglion.

Loss of taste in the anterior two-thirds of the tongue may be present but is usually not total and may eventually return (p. 648).

Trophic, vasomotor, and secretory symptoms are prominent. At first, due to irritation, there may be increased salivary and lacrimal secretion, but as destruction occurs there is dryness of the mucous membrane of the affected side, frequently ulceration and destruction of the cornea, and pallor of the face. *Herpes* may occur as an early symptom.

The *motor symptoms* if present are paralysis of the muscles of mastication (p. 565). Atrophy of the muscles and tissues of one side of the face may occur from disease of this nerve (*progressive facial hemiatrophy*). This usually appears before puberty and shows itself first by a thinning of the skin. Later the bones, cartilage and muscles are affected. There is no paralysis or reaction of degeneration. One-half of the tongue may be atrophied but it is protruded straight. In atrophy due to paralysis it is usually protruded toward the paralyzed side.

Degeneration of the descending root, neuritis of the nerve, and inflammation of the Gasserian ganglion have been found in those suffering from these symptoms. It may occur in syringomyelia (See also p. 807).

Spasm of the muscles of mastication occurs as an early symptom in tetanus, and in mild cases may be confined to these muscles.

Neuralgia of the fifth nerve is described on page 574.

2. *Seventh or Facial Nerve*

The cortical center of this nerve is located in the lower part of the precentral convolution. From these cells fibers converge to the anterior portion of the posterior limb of the internal capsule. From there they pass through the pons, where they decussate and join the nucleus of the opposite side (Fig. 137). From the nucleus the fibers take a circuitous course, bending around the sixth nerve nucleus (Fig. 147) and emerge at the lower part of the pons. Here the auditory nerve is separated but very slightly from it (Fig. 148).

The symptoms of destructive disease of the nerve differ somewhat according as the lesion is in one of three subdivisions: (1) an intracranial portion, extending from the superficial origin to the internal auditory meatus, (2) from the internal auditory meatus through the facial canal to the stylomastoid foramen, (3) after it emerges from the stylomastoid foramen.

Within the facial canal, at what is known as the knee, is situated the geniculate ganglion. From this arise two nerves, the chorda tympani, running peripherally with the trunk of the facial to join the lingual branch of the fifth, and the intermediary nerve of Wrisberg, which runs centrally (p. 648).

The seventh nerve supplies the muscles of the face, eyelids, and mouth, excepting the elevator of the upper lid and the muscles of mastication; also the occipitofrontalis, platysma, muscles of the eyebrows, extrinsic muscles of the ear and nose, posterior belly of the digastric and the stylohyoid. It conveys sensations of pressure, and through the chorda tympani, sensations of taste from the anterior two-thirds of the tongue (Fig. 152), and through this nerve sends secretory fibers to the salivary glands.

While with the exceptions above noted, the seventh is usually regarded as a pure motor nerve, Hunt (J. Nerv. & Ment. Dis., Feb., 1907, p. 73) believes that it supplies the internal part of the auricle and the external auditory canal with sensory fibers. If such is the case the geniculate ganglion corresponds to the ganglia found on the sensory roots (p. 556) and the *pars intermedia* of Wrisberg is the afferent division.

Paralysis of the functions of the facial nerve may be due to a lesion of either the central or peripheral neuron.

Central or supranuclear paralysis (lesion involving cortical cells or fibers running from them to the nucleus) may be caused by either a vascular lesion (apoplexy), tumor, abscess, inflammation of cortical cells (encephalitis) or patch of sclerosis (multiple sclerosis).

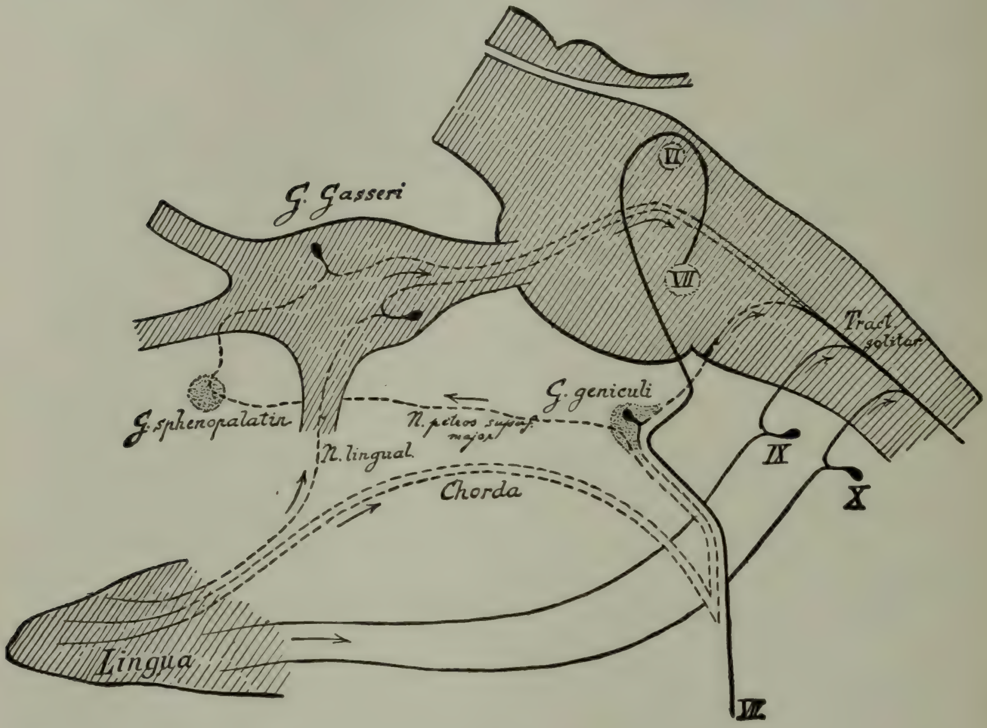

Fig. 152.—Possible Conduction Paths for Gustatory Impulses. (After Villiger's "Brain and Spinal Cord" (Piersol Translation), published by J. B. Lippincott Co., Philadelphia.)

Paralysis of the muscles when expressing emotions only may be due to lesions of the optic thalamus.

In supranuclear palsy the weakness is confined to the muscles about the angle of the mouth, those of the upper part of the face escaping. Unless the lesion is in or near the cortex, paralysis of the arm and leg are also present. This is due to the fact that after the nerve fibers leave the cortical cells they converge into a small space and it is impossible for a lesion to cause involvement of some fibers without involving all (Fig. 137).

Why the orbicularis palpebrarum and muscles of the forehead and eyebrows escape is not definitely known. It may possibly be due to the well-known fact that muscles which habitually act together may be innervated from either side of the brain. These muscles are among those which usually act on both sides at the same time. This is further borne out by the fact that at times, in the early stages of an acute paralysis due to a central lesion (apoplexy), the upper part of the face may be involved for a short time.

Peripheral paralysis may be due to a lesion either of the nucleus or nerve trunk.

Nuclear palsy is usually due to a progressive nuclear degeneration and is associated with a similar condition of the nuclei of the ninth, tenth, eleventh and twelfth nerves (bulbar palsy).

In such cases the paralysis is *incomplete,* owing to the fact that the nucleus is destroyed slowly, and hence for a long time there are some healthy cells, is bilateral and will show evidences of the reaction of degeneration.

Acute nuclear disease may also occur due to either the toxins of the infectious fevers, especially poliomyelitis, alcoholic or mineral poison, hemorrhage from some branch of the vertebrals supplying the medulla or thrombosis of the posterior inferior cerebellar artery (p. 712). When inflammatory in origin, it is known as *polioencephalitis inferior.* When due to toxemia the weakness is usually bilateral; if due to a vascular lesion it is unilateral.

Tumor or an apoplectic disturbance in the pons may involve the nucleus of one side with the motor, and, possibly, sensory tracts. In such a case there would be a *complete* facial paralysis on the side of the lesion with that of the arm and leg on the opposite side (decussation in the medulla) (Fig. 137).

Disease of the nerve trunk is the most common cause of facial paralysis. If the *first or intracranial division* (*supra*) is the seat of the lesion it may be either a tumor or meningitic exudation at the base (cerebellopontile angle) or fracture of the base (posterior fossa).

In these cases the paralysis is *complete,* the patient being unable to either close the eye or wrinkle the forehead on the affected side.

Owing to relaxation of the orbicularis palpebrarum the lower lid droops and the tears flow over the cheek. Food is apt to collect between the gums and cheek, owing to the weakness of the buccinator. Reaction of degeneration is present in the affected muscles.

There is no loss of taste, and other cranial nerves, especially the eighth, are usually affected also (p. 641). If the sixth is involved there is not loss of conjugate lateral movement of the eyes (p. 636). If the lesion is in the cerebropontile angle cerebellar symptoms are usually present. A history of traumatism will indicate fracture at the base of the skull.

The most common location of the lesion is within the facial canal, the second portion of the nerve (*supra*) being involved, and is commonly known as *Bell's palsy.*

Causes.—The causes of trouble here in the order of frequency are: exposure to cold, especially if the side of the face has been exposed to a draft of cold air (refrigeration palsy); extension of inflammation from the middle ear; after operations on the mastoid and middle ear; arteriosclerosis.

The **symptoms** are similar to those described as due to lesions of the first part with the addition of loss of taste on the affected side in the anterior two-thirds of the tongue, owing to involvement of the chorda tympani (p. 641); sometimes pain over the mastoid or within the ear and the herpes of the auricle.

Contractures and muscular twitchings may eventually develop in the affected muscles, causing drawing of the angle of the mouth to the paralyzed side.

Other cranial nerves are not affected unless inflammation of the middle ear or mastoid is present, when symptoms of these conditions will be found.

When the paralysis occurs in old people with arteriosclerosis and no other cause is apparent, it may be due to lack of blood supply in the nerve due to that condition.

Lesion of the third part (*supra*) may be due to pressure of the forceps during labor, implication in cellulitis or growths of the neck, disease of the parotid gland, and infectious diseases.

The **symptoms** are similar to those due to a lesion in the other two portions, except taste is not lost, no other nerves are affected, and some of the branches of the nerve may escape injury since the paralysis sometimes may be incomplete.

Bilateral palsy rarely occurs. It may be due to basal meningitis, usually syphilitic; as a part of multiple neuritis, usually either diphtheritic or alcoholic; bilateral otitis media.

Spasm of the Muscles Supplied by the Seventh Nerve.—This is variously known as *painless tic, histrionic spasm, mimic spasm, and myospasm.*[1] It may be clonic or tonic, usually the former.

The causes may be an irritating lesion, as either tumor, meningitis, hemorrhage, or abscess involving the cortical center, for the facial muscles (Jacksonian epilepsy, p. 614); reflexly from irritation of a branch of the trigeminal nerve, as by a decayed tooth or during a paroxysm of tic douloureux; reflexly from irritation of the ocular nerves, as eye strain, foreign bodies in the eye, inflammatory affections; hysteria; overwork, worry or fatigue; anything which causes chronic ill health; cases in which no cause can be found, but probably due to some functional disorder of the cortical centers.

When confined to the orbicularis palpebrarum, as it often is, especially in eye conditions, it is termed *blepharospasm* or *nictitating spasm. Tonic spasm* due to secondary contractures may follow Bell's palsy (p. 643).

Spasm must be distinguished from *tic* (p. 764). If *hysterical,* other symptoms will be present (*vide*). If due to *organic disease*, paralysis and other symptoms of the condition will sooner or later be manifest.

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[1] This latter term may be applied to any spasm.

3. Eighth or Auditory Nerve

This nerve is divided into two parts: the cochlear and vestibular. The former has to do with hearing, the cortical center for which is in the first temporal convolution (Fig. 135). The latter is more important to the neurologist, its peripheral ending being in the semicircular canals. It has close connection with the cerebellum and posterior longitudinal fasciculus which unites the different nuclei of the nerves which move the eyeball (p. 633). Its function is to maintain equilibrium and our relations with space; in this it is assisted by the extra-ocular muscles and joints.

The auditory nerve tract may be affected by middle or internal ear disease; lesions at the base of the brain as either meningitis (cerebrospinal or syphilitic) or tumor (cerebellopontile angle, p. 722); degeneration of the nerve or nuclei in tabes; lesions involving the nuclei connected with the nerves in the upper part of the medulla (p. 583); the tegmentum (posterior bodies of the corpora quadrigemina), internal geniculate body, when the symptoms will be on the opposite side, and the temporal lobes; if on the left side, word deafness results (p. 617). To cause total deafness both sides must be affected. Cases in which there was an acute development of deafness, vertigo, and paralysis of other cranial nerves, especially the seventh, have been ascribed to a toxic neuritis of the auditory nerve. Such cases would differ from the symptoms caused by disease in the cerebellopontile angle by their sudden onset.

Symptoms.—The symptoms referable to the auditory nerve are deafness, tinnitus and vertigo. The tests to determine if *deafness* is due to lesion of the middle ear or to disease of the internal ear or nerve are given on page 602. If the nerve between the base and internal auditory meatus is involved the facial is usually also affected. Care must be taken in such cases to exclude disease of the middle ear.

Tinnitus, or ringing in the ears, indicates irritation of some part of the auditory apparatus. It may be due to something in the external auditory canal, as impacted wax; to middle ear disease; to disease of the internal ear and disease of the nerve itself (p. 647).

It may be either *continuous* or *pulsating.* If the former is increased by the recumbent position it is due to congestion; if relieved, it is due to anemia. Nitrite of amyl aggravates the former and relieves the latter. It may be due to certain drugs, especially quinin and salicylic acid in some form. If pulsating tinnitus is synchronous with the pulse, it may be due to intracranial aneurism, but may occur in neurasthenia. Clicking sounds may be due to clonic spasm of the tensor tympani muscle. Irritative lesions, as a tumor affecting the centers in the temporal lobe, may cause various kinds of hallucinations of hearing (voices, etc.).

Vertigo, or giddiness, is the consciousness of disturbed equilibrium due to a disturbance of the nervous mechanism which governs the relation of the body to external objects. For securing the balance or equilibrium of the body in its continually changing relations with external objects, accurately timed and ever changing muscular contractions are necessary; in other words, muscle synergy (p. 581).

The motor impulses necessary to cause these contractions are determined in the cortical centers by sensory impressions received from the eye and its muscles, semicircular canals of the internal ear, from the skin of those parts which may be in contact with external objects, and from the articular surfaces and muscles controlling them. These impressions go to the cerebellum, the organ which controls muscle synergy, and anything which disturbs the transmission or reception of these impressions causes a derangement of the synergy of the motor impulses which is manifested by vertigo (*infra*). The nausea, vomiting, and irregular pulse which may coexist, are due to reflex irritation of the pneumogastric nerve.

Causes.—The causes are:

1. Visual defects, as errors in refraction and loss of muscle balance.

2. Aural disturbances, as either disease of the labyrinth (Ménière's disease), or conditions irritating it, as impacted wax, middle ear disease, pressure of the stapes upon the fluid in the labyrinth (p. 647).

3. Toxemia, as alcohol, tobacco, quinin, salicylic acid, auto-intoxication due to constipation or digestive disorder.

4. Derangement of the cerebral circulation causing either cerebral hyperemia or anemia. The former type may occur in mitral disease and in women at the time of the menopause, the latter in aortic disease, fatty heart and arteriosclerosis, when it may be a premonitory sign of oncoming thrombosis (p. 711).

5. Neuroses, as neurasthenia and hysteria.

6. Organic disease of the brain, as multiple sclerosis and brain tumor, especially if cerebellar. Tumor anywhere in the brain may cause vertigo by changing the pressure within the skull. A lesion of the cerebellum is especially liable to do this (p. 581). *Intracerebellar tumors* cause a vertigo in which the sense of rotation of the body is in the same direction as that of the apparent movement of external objects (away from the side of the lesion). In *extracerebellar tumors,* while external objects appear to move as above, the subjective sensation of rotation is the reverse, i. e., towards the lesion.

7. Obstruction of the nasal passages and inflammation or irritation of the ethmoidal and frontal sinuses.

8. Acute giddiness may be the aura of an epileptic fit or due to sea sickness, swinging, rapid rotation of the body, blows on the head, looking down from great heights, and passing a constant electric current through the head.

9. A hereditary form of vertigo has been described.

Vertigo may be *subjective or objective.* In the former the patient feels as if he were whirling rapidly about, in the latter objects seem either to whirl about the patient or to move up or down.

The attack usually comes on suddenly and may be brought about by suddenly rising from a sitting or recumbent position, by turning the head in some certain direction, or jarring the body. It may last a short time or for several hours. Mental confusion, nystagmus, nausea, vomiting, pallor and irregular pulse may be associated.

Ménière's disease is due probably to hemorrhage into or inflammation of the labyrinth. This may be due to previous traumatism, gout, or syphilis. It occurs after middle life and is characterized by sudden and complete deafness, nausea, vomiting, tinnitus, spontaneous nystagmus (p. 583) and intense vertigo occurring in a person who has not had previous ear disease. In the attack the patient may fall and may be unconscious. After the attack the gait may be staggering, and remain so for days or years. Bone conduction is diminished or lost on the affected side.

This condition must not be confounded with *Ménière's symptom complex,* a condition in which there are periodical attacks of the symptoms described above, but in which there is a history of previous deafness (not complete) and ear disease, which did not develop suddenly. The symptoms are not so severe as those of true Ménière's disease and are usually due to either an intermittent closure of the Eustachian tubes or a temporary congestion of or exudation into the labyrinth due to middle ear disease. The former causes retraction of the membrana tympani and forcing of the stapes into the oval window which increases the tension of the labyrinthine fluids. Inflation of the tympanic cavity usually relieves the symptoms.

These conditions may be confounded with *epilepsy.* Differentiation should not be difficult if convulsions occur, as these are not symptoms of aural vertigo. Epileptic attacks may occur without convulsion and these may be mistaken for attacks of severe vertigo in which the patient falls. In the former tinnitus, deafness, evidence of disease in the middle and internal ear, forced movements and irregular gait, are not present, while in the latter they are. Consciousness is not usually lost in vertigo, but always is in epilepsy. Vertigo of this type is rare in the young, while epilepsy is common.

4. Ninth or Glossopharyngeal Nerve

This nerve has three functions: motor, common sensibility and special sense, viz., taste.

The nerve probably supplies the upper pharyngeal muscles with motor fibers and the mucous membrane of the pharynx, and the fauces, tonsils and tympanic cavity with sensory fibers. The nuclei and cortical centers for these functions are in common with those of the tenth nerve, *Sensations of taste* from the posterior third of the tongue reach the nucleus of the tractus solitarius in the medulla by this nerve. From the anterior two-thirds there is some dispute as to the exact route: the most generally accepted view is that from the lingual nerve sensations pass through the chorda tympani to the geniculate ganglion and from there by the intermediary nerve of Wrisberg to the medulla, where they form a descending root which joins the tractus solitarius.

The view, that these sensations pass to the Gasserian ganglion, is doubtful, as removal of the ganglion does not cause permanent loss of taste (Fig. 152). From the nucleus of the tractus solitarius they pass to the optic thalamus and from there to the uncinate gyrus (Fig. 136).

The *motor symptoms* referred to this nerve are usually associated with symptoms of disease of the seventh, tenth, eleventh and twelfth nerves (See Bulbar Palsy). The nerve trunk may be involved at the base of the brain in meningitis or tumor. If the nerve trunk is involved, in addition to interference with swallowing, there is loss of the sense of taste in the posterior third of the tongue (For examination of this sense, see p. 602), anesthesia of the pharynx and loss of the pharyngeal or palatal reflex (p. 589).

Loss of taste is known as *ageusia;* if more acute than normal, *hypergeusia;* and if perverted, *parageusia.*

Parageusia may occur as an epileptic aura, especially in organic lesions of the uncinate gyrus (p. 768). Ageusia, parageusia and anesthesia of the pharynx may be symptoms of hysteria (For symptoms of spasm, see pp. 650 and 780).

5. *Tenth or Pneumogastric or Vagus Nerve*

This nerve has motor and sensory functions. The *course of the motor fibers* is as follows:

The central neuron—from the cerebral cortex (foot of ascending frontal convolution through the internal capsule to the nucleus ambiguus and nucleus vagi et glossopharyngei in the floor of the fourth ventricle.

The peripheral neuron consists of these nuclei and of the nerve fibers arising from them which constitute the ninth and tenth nerves. From the former the eleventh nerve arises also. The fibers of the former supply voluntary muscles, while those from the latter form part of the vegetative system (p. 594).

The sensory fibers rise from the superior or jugular and the inferior or petrous ganglia in the case of the ninth, and the ganglia of the root or jugular ganglia and the ganglia of the trunk, or nodular ganglia, in the case of the tenth nerve.

These ganglia are situated on the nerve trunks near the jugular foramen. From the cells of these ganglia the axons go to the tractus solitarius and its nucleus and the nucleus alæ cinereæ. The dendrites form the peripheral nerves (p. 556). From the nuclei fibers pass through the medial fillet to the optic thalamus and from there to the cortex.

These nerves, together with the spinal accessory, have connections with the cerebellum, fifth nerve and sympathetic system.

The tenth nerve is the *motor nerve* for the laryngeal and many of the pharyngeal muscles; the *sensory nerve* for the larynx, part of the pharynx and thoracic and abdominal viscera; the *inhibitory nerve* of the heart; the *motor nerve* of the esophagus and partly of the stomach, and has *vasodilator and secretory functions.* It is part of the *autonomic system* (p. 594) (See also the eleventh nerve).

Paralysis of the tenth nerve is most commonly due to degeneration of the nucleii in the disease known as chronic bulbar palsy. Acute paralysis may be due to hemorrhage into the medulla or to inflammation following infectious fevers, of which acute poliomyelitis is the most common (polioencephalitis inferior). At its peripheral origin (Fig. 148) it may be damaged by basal meningitis due to various causes, or by tumor or aneurism of the vertebral artery. Nuclear paralysis is usually bilateral; if due to disease of the nerve trunk it is usually unilateral.

Disease of the cortical center may cause laryngeal and pharyngeal paralysis, but as these muscles are innervated from either side, it soon disappears unless the lesion is bilateral (p. 708).

The inferior or recurrent laryngeal branch is affected by a number of different causes. It is the motor nerve of the larynx, supplying all the muscles but the cricothyroid, this being supplied by the superior laryngeal.

In a discussion of unilateral paralysis of this nerve (Proc. Royal Soc. Med., June, 1913, Laryngol. Section, p. 139) Hall presented the following table of its causes and their relative frequency:

	Right	Left
Aneurism of arch of aorta	3	28
Aneurism of arch of aorta (subclavian)	1	..
Mitral stenosis and enlarged left auricle	0	4
Enlarged bronchial and other glands	1	6
Disease of apex of lung	1	0
Malignant disease of lung	0	8
New growths in thorax	0	5
Malignant disease of esophagus	9	8
Thyroid tumors	3	2
Influenza (vocal cord not stated, 1)	2	0
Diphtheria, lead (1 each)	0	2
Neuritis	4	2
Tabes, syringomyelia, multiple sclerosis, bulbar palsy, paresis	3	6
In combination with other paralyses, as hemiplegia, facial, palate, pharynx, trapezius and sternomastoid	6	4
Doubtful cause	0	7
	33	82

It is likely that the lesion in those caused by influenza, diphtheria and lead was neuritis. When occurring with organic diseases of the nervous system it is degeneration, and when complicating visceral diseases the paralysis is due to pressure. In addition to the causes above mentioned, laryngeal palsy may be due to hysteria, weakness from anemia or exhaustion, congestion of and tumors of the larynx.

The various forms and symptoms of laryngeal paralysis are given in the following table from Gowers:

Symptoms	Signs	Lesion
No voice; no cough; stridor only on deep inspiration	Both cords moderately abducted and motionless	Total bilateral palsy
Voice low pitched and hoarse; no cough; stridor absent or slight on deep breathing	One cord moderately abducted and motionless; the other moving freely, and even beyond the middle line in phonation	Total unilateral palsy
Voice little changed; cough normal; inspiration difficult and long, with loud stridor	Both cords near together, and during inspiration not separated, but even drawn nearer together	Total abductor palsy
Symptoms inconclusive; little affection of voice or cough	One cord near the middle line not moving during inspiration, the other normal	Unilateral abductor palsy
No voice; perfect cough; no stridor nor dyspnea	Cords normal in position and moving normally in respiration; but not brought together on an attempt at phonation	Abductor palsy

Organic disease attacks the abductors first, while in functional disease, as hysteria, the adductors suffer first and usually are the only muscles affected.

Spasm of the laryngeal muscles most commonly occurs in rachitic children, and is known as spasmodic croup. If it occurs in adults it is usually due to hysteria. It may be due to irritation of the nerve somewhere in its course, and is a symptom of tetany (laryngismus stridulus).

The laryngeal crises of tabes dorsalis are probably due to spasm of the adductors.

Anesthesia of the larynx may be due to hysteria or to organic lesion. If the former, reflex action is preserved; if the latter, it is not.

Paralysis of the pharyngeal branches causes difficulty in swallowing; this symptom is only marked in bilateral lesions. *Spasm* causes temporary inability to swallow and a feeling as if a ball were in the throat. It is usually due to hysteria (globus hystericus).

Paralysis of the esophageal branches is rare and occasions trouble in the food reaching the stomach. *Spasm* is more common and may simulate organic stricture of the esophagus; it is usually due to hysteria. When so caused it will relax under an anesthetic, so that a bougie can be readily passed.

Gastralgia may be due to *irritation of the gastric branches.* The gastric crises of tabes dorsalis are also so caused.

Irritation of the cardiac branches causes slowing of the heart (bradycardia). Bronchial asthma and possibly hiccough may be due to *irritation of the pulmonary branches. Paralysis* causes rapidity of the heart (tachycardia), often occurring after diphtheria (diphtheritic neuritis). If unilateral, not much disturbance may occur. Palpitation of the heart may be, and pseudo-angina pectoris is due to *irritation of these branches.*

Irritation of the nerve trunk in the neck may cause bradycardia, vomiting, and spasm of the laryngeal muscles.

The *symptoms of paralysis* are tachycardia and weakness of the pharyngeal and laryngeal muscles (See also Vagotonia, p. 595).

6. *Eleventh or Spinal Accessory Nerve*

This nerve consists of two parts, the *accessory part* to the vagus and the *spinal portion.*

The *accessory portion* arises from a nucleus in the medulla just below the nucleus ambiguus and connects with it. It extends downward as far as the intermediolateral tract (p. 593). The nerve fibers arising from it join the vagus and are probably distributed to the laryngeal and pharyngeal muscles. The latter arises in the cervical region of the cord in the intermediolateral tract. The nerve trunk ascends through the foramen magnum into the cranial cavity, runs with the vagus through the jugular foramen into the neck and supplies the sternocleidomastoid and trapezius muscles.

Paralysis of the spinal portion may be due to basal meningitis from any cause or the gray matter from which it arises may be the seat of either hemorrhage, acute poliomyelitis or chronic degeneration (progressive spinal muscular atrophy). It may be affected in caries of the cervical vertebra, in tumors of the neck, and by neuritis. The latter may be due to pressure from heavy weights carried on the shoulder.

The symptoms, if unilateral, consist of difficulty in turning the head to the opposite side, drooping of the shoulder and impairment of the power of elevating the arm above a right angle with the body. If bilateral, the head is without support and drops forward on the body. If due to basal meningitis, symptoms of that disease are present and other nerves are usually affected.

Cervical caries is accompanied by pain on jarring, rigidity of the neck, tenderness, possibly kyphosis, and the x-ray will show evidences of bone disease.

The symptoms of acute poliomyelitis (p. 188) or chronic progressive spinal atrophy (p. 694) will be present if it is part of those diseases. Hemorrhage will be sudden in onset and probably follow trauma (p. 727).

Spasm of the muscles supplied by the spinal portion may be acute or chronic, tonic or clonic. It is known as *torticollis, or wry neck.* It may be due to *organic or functional causes.* The former may be either irritation of the nerve at the base of the brain or in the neck, as meningitis, cervical pachymeningitis, intra- or extraspinal tumor, cervical caries, or irritation of any of the nerves which anastomose with it (upper cervical roots), or focal lesion in the cortical centers. In cervical lesion the spasm is apt to be tonic; in cortical lesion it is usually clonic (Jacksonian epilepsy).

The functional type is probably more common and is of doubtful etiology. It occurs in those who are neurotic and have been overworked or subjected to other undue strain. Eye strain has been given·as a cause. It may be hysterical and also one of the manifestations of tic (p. 764). To some peculiar functional derangement of the cortical centers most cases are attributed.

The symptoms, no matter what the cause, are, if tonic and unilateral, a rotation of the head, so that the chin points upwards and toward the opposite side, the occiput being drawn toward the shoulder of the side affected. When the trapezius alone is affected the head is drawn backward and the shoulder elevated. In the clonic form the head is thrown into the above position at regular intervals and contraction of the muscles will be observed. The posterior rotators of the opposite side, supplied by the upper cervical nerves, are usually also involved; if they are the head is pulled back more than in pure spinal accessory spasm. Muscles supplied by other nerves (face, shoulder, arm) may eventually be affected. The movements in functional cases disappear during sleep. The muscles may become hypertrophied.

Bilateral spasm of the sternomastoid pulls the head forward and downward. Young children are rarely met who at regular intervals nod the head (eclampsia nutans). It is due to bilateral clonic sternomastoid spasm and the victims are usually rachitic.

Sternomastoid spasm must be distinguished from that of the *splenius capitis* of the other side. In this the head is drawn backward toward the affected side, and the chin is depressed and directed toward the corresponding shoulder. In spasm of the *obliquus capitis inferior* the head is rotated without either elevation or depression of the chin. These muscles may be affected in association with spinal accessory spasm.

It must be determined if any of the organic causes mentioned are present before deciding that the condition is functional.

7. *Twelfth or Hypoglossal Nerve.*

The cortical center is situated in the lower part of the ascending frontal convolution. The fibers from here run in association with the pyramidal tract through the internal capsule to the nucleus in the floor of the fourth ventricle in the lower part of the medulla. From here the nerve arises (Fig. 147). It is the motor nerve of the tongue, supplying all its muscles except the palatoglossus and pharyngoglossus. It has connections with the tenth, lingual, upper three cervical and sympathetic nerves.

Paralysis may be due to a lesion *above the nucleus* (supranuclear), which usually occurs as part of a hemiplegia due to apoplexy, tumor, or abscess, anywhere from the cortex to the nucleus.

Nuclear disease is due to acute inflammation (polioencephalitis), hemorrhage into the medulla, or chronic degeneration (bulbar palsy). The *nerve trunk* may be affected by basal meningitis from any cause, fracture or tumor at the base, penetrating wounds or tumors high up in the neck.

The symptoms, when unilateral, are protrusion of the tongue toward the paralyzed side, it being pushed around by the normal muscles. Occasionally there is protrusion away from the paralyzed side.

In *bilateral paralysis* the tongue can only be protruded slightly or not at all. In supranuclear or central neuron lesions there is no atrophy, in disease of the nucleus or nerve trunk (peripheral neuron) there is (Fig. 174), the tongue being wrinkled and tremulous. *Crossed paralysis,* the tongue on one side and the face, arm and leg on the other, is due to a lesion in the medulla near the nucleus. If due to lesion at the base of the brain, other cranial nerves will probably be affected and symptoms of the causative lesion will be present. This is not a common condition (See Chronic Bulbar Palsy).

Combined paralysis of the motor branch of the fifth, seventh, ninth, tenth, eleventh and twelfth nerves, due to either inflammation or degeneration of their nuclei, occurs. It is know as polioencephalitis inferior, either acute or chronic (See p. 692).

2. Spinal Nerves

(*a*) *Cervical Nerves*

The branches arising from the posterior divisions of the upper four may be the seat of pain (See Cervico-occipital Neuralgia, p. 575).

Paralysis of the phrenic nerve, which is derived from the anterior divisions of the third, fourth, and fifth cervical nerves, may be due to either hemorrhage into, tumor, or inflammation of the cord in the region of these segments; to cervical meningitis; to vertebral caries, to wounds in the neck, compression by tumor or aneurism in the neck or thorax, and the nerve may be affected with neuritis either separately or with other nerves in multiple neuritis.

When both nerves are affected there is inaction of the diaphragm, shown by the upper part of the abdomen not advancing, and sometimes being retracted during inspiration. Dyspnea will be present. Involvement of one nerve does not cause marked symptoms.

Disease of either the cord, meninges, or vertebra is usually the cause of bilateral palsy, and more or less paralysis below the seat of the lesion would probably be present (See Myelitis; Tumor of Cord). Inaction of the diaphragm may be due to either peritonitis, either diaphragmatic or subdiaphragmatic pleurisy, owing to the pain caused by such movement.

Hiccough is believed to be due to spasm of the diaphragm from irritation of this nerve.

Brachial Plexus

This is composed of the anterior divisions of the fifth, sixth, seventh, and eighth cervical and first dorsal nerve (Fig. 153).

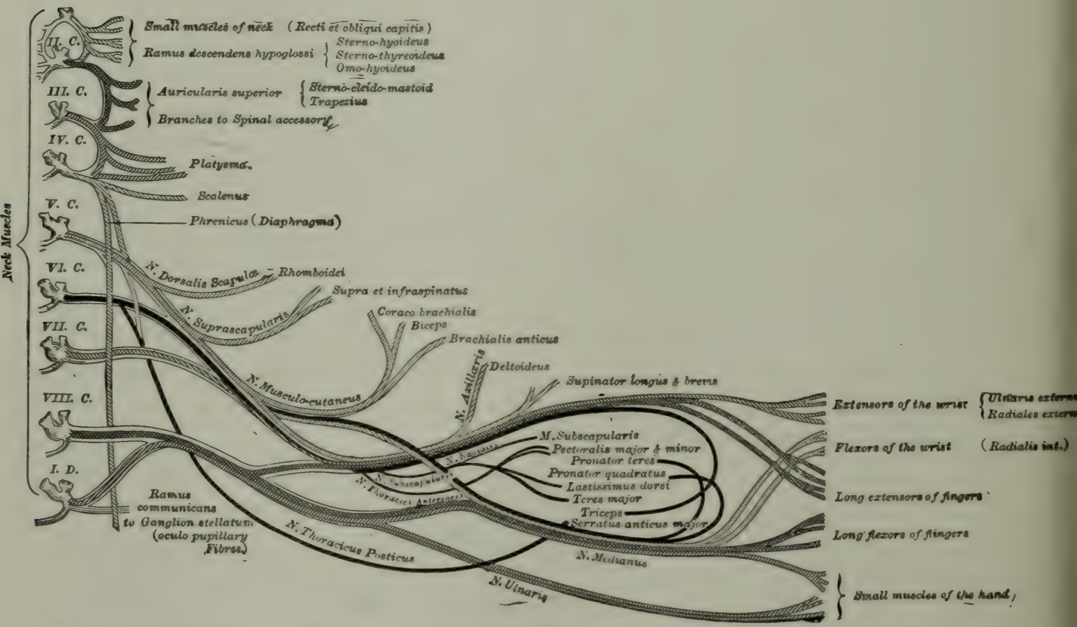

Fig. 153.—The Cervicobrachial Plexus and Its Branches. (After Kocher: from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

In general, it may be said that paralysis of the muscles supplied by these nerves may be due to compression; neuritis, traumatic or toxic; acute poliomyelitis; progressive spinal muscular atrophy; disease of the gray matter of the cervical region of the cord (myelitis, tumor, syringomyelia, hemorrhage); pachymeningitis; and dystrophy. Some of these causes are more liable to affect the muscles supplied by certain nerves than others. For doubtful cases the reader is referred to the description of the various diseases mentioned.

1. Posterior Thoracic Nerve

This nerve arises from the fifth and sixth roots and supplies the serratus magnus muscle. It may suffer from paralysis due to neuritis caused by either a blow on the neck, exposure to cold, toxemia, pressure from either a weight carried on the shoulder or violent muscular effort, as in lifting a heavy weight.

The **symptoms** are: (1) rotation of the scapula upon its vertical axis when the arm is thrown forward, with recession of the edge of the scapula from the thorax ("winged scapula"); (2) rotation upward and inward of the lower angle of the scapula when the arm is advanced; (3) weakening or loss of the power of elevating the arm above the shoulder (Fig. 158).

2. Suprascapular Nerve

This nerve also arises from the fifth and sixth roots (Fig. 153). It supplies the infra- and supraspinatus muscles.

Paralysis causes loss of the power of outward rotation of the humerus, shown by inability to carry the hand from left to right in writing. It is usually found associated with paralysis of other muscles, due to one of the causes mentioned on page 654, but most frequently associated with paralysis of the deltoid, due to injury by dislocation of the shoulder.

3. Circumflex Nerve

This arises from the posterior cord of the plexus (Fig. 153). It supplies the deltoid and teres minor muscles. *Paralysis* is most frequently due to its injury by either blows or falls on the shoulder or dislocation of the shoulder joint.

The **symptoms** are loss of power of elevating the arm from the body. More or less atrophy of the muscle occurs with change in the electrical reactions (p. 610). Adhesions in the joint may form.

Care must be taken not to confuse loss of motion due to paralysis with that due to ankylosis from arthritis. In the latter, symptoms of joint inflammation will have preceded the disability and the joint will be fixed. It must also be borne in mind that atrophy and weakness of muscles in the neighborhood of a diseased joint may occur which are greater than that which would occur from disuse (p. 676). There may be *loss of sensation* over the deltoid (*ax* in Figs. 154 and 155).

4. *Musculocutaneous Nerve*

This nerve arises from the outer cord (Fig. 153). It supplies the biceps and brachialis anticus muscles.

Paralysis causes loss of power of flexing the forearm on the arm, most marked when the forearm is supinated and the supinator longus cannot act as a flexor. If sensory loss is present, it is found in the radial side of the forearm (*cl* in Figs. 154, 155).

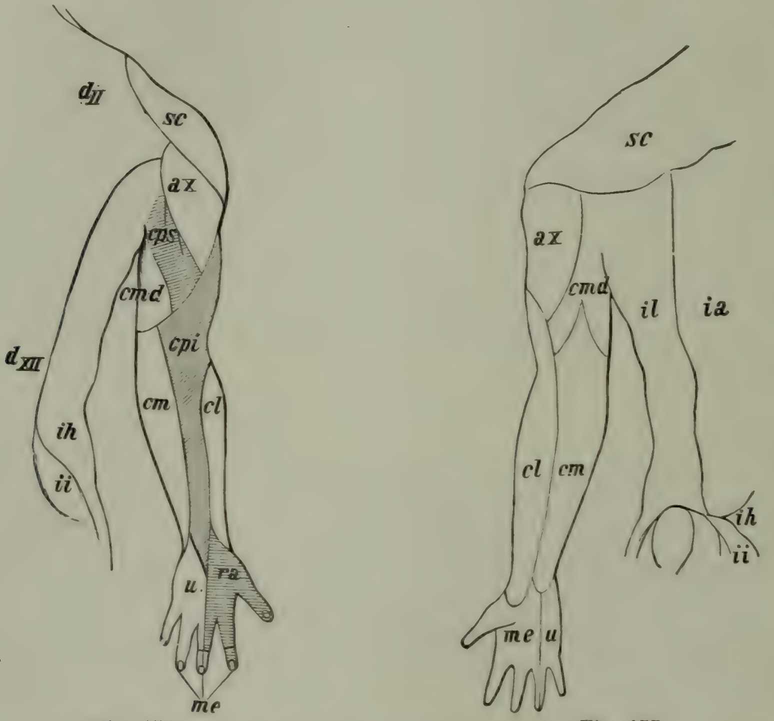

Fig. 154.

Fig. 155.

Figs. 154 and 155.—Distribution of the Sensory Nerves in the Trunk and Upper Extremities. Fig. 154, Posterior Aspect. Fig. 155, Anterior Aspect. The Shaded Portion in Fig. 154 Designates the Territory Supplied by the Radial Nerve. (From Henle.) *sc*, Supraclavicular Nerves (from the Cervical Plexus); *ax*, Cutaneous Branch of the Axillary Nerve; *cps* and *cpi*, Superior and Inferior Posterior Cutaneous Nerves from the Radial (*ra*); *cmd*, *cm*, and *cl*, Median Cutaneous, Median, and Lateral Nerves; *me*, Median Nerve; *u*, Ulnar Nerve; *d* II, Second Dorsal Nerve; *d* XII, Twelfth Dorsal Nerve; *ih*, Iliohypogastric Nerve; *ii*, Ilio-inguinal Nerve; Lateral Perforating Branches, and *iam*, Anterior Perforating Branches of the Intercostal Nerves. (After Strümpell.)

5. *Musculospiral Nerve*

This nerve is derived from the posterior cord of the brachial plexus (Fig. 153). It supplies the triceps, anconeus, supinator longus, extensor carpi radialis longior, brachialis anticus, and through its posterior interosseus branch, all of the extensor muscles of the radial and posterior brachial region.

In *paralysis* due to a lesion above the origin of the posterior interosseus there is loss of extension of the forearm upon the arm, of extension of the hand upon the forearm, and impairment of supination. The fingers are flexed at the distal joints and the grip is apparently weakened, owing to loss of resistance of the extensors (p. 564). If long continued, excessive flexion leads to undue prominence of the carpal bones and synovial sacs at the back of the wrist (Fig. 157).

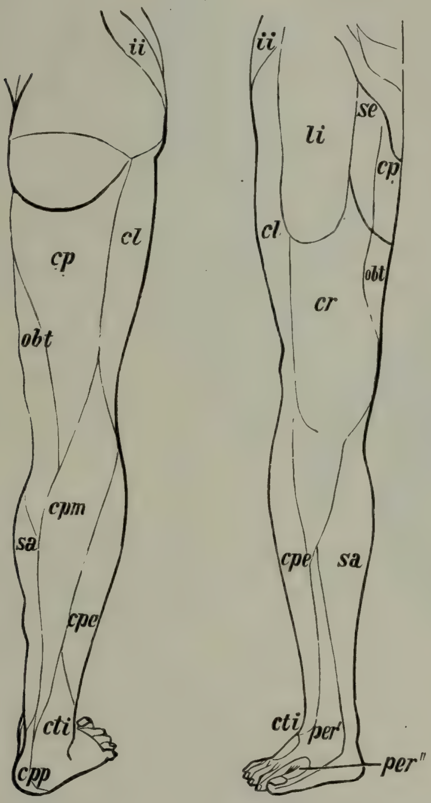

Fig. 156. Fig. 156a.

Figs. 156 and 156a.—Distribution of the Sensory Cutaneous Nerves to the Lower Extremities. Fig. 156, Posterior Aspect. Fig. 156a, Anterior Aspect. (From Henle.) *ii,* Ilio-inguinal Nerve; *li,* Lumbo-inguinal Nerve; *se,* External Spermatic; *cp,* Posterior Cutaneous; *cl,* Lateral Cutaneous; *cr,* Crural; *obt,* Obturator; *sa,* Saphenous; *cpe,* Peroneal Communicating Nerve; *cti,* Tibial Communicating Nerve; *per',* Superficial Branch of the Peroneal Nerve; *per'',* Deep Peroneal Nerve; *cpm,* Posterior Median Cutaneous Nerve; *cpp,* Cutaneous Plantar Nerve. (After Strümpell.)

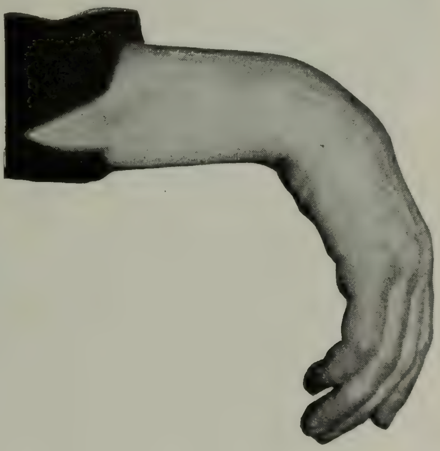

Fig. 157.—Dropped Wrist from Musculospiral Palsy, Showing Retrocarpal Tumor. (After Church and Peterson's "Nervous and Mental Diseases," published by W. B. Saunders Co., Philadelphia.)

If only the posterior interosseus branch is affected, the triceps and supinator longus escape. Atrophy of the affected muscle and changes in the electrical reactions (p. 610) may be present. Loss of sensation, if present, is confined usually to the dorsal surface of the thumb and of the hand, corresponding to the index and middle fingers and radial side of the forearm (Figs. 154 and 159). This nerve is frequently paralyzed by pressure (p. 667), traumatism, as fracture of the humerus, and stretching from over-elevation of the arm.

6. *Median Nerve*

The median nerve arises from the outer and inner cords of the plexus (Fig. 153). It supplies the pronators, flexor carpi radialis, two outer lumbricals, all the muscles of the ball of the thumb, the abductor pollicis, outer half of the flexor brevis pollicis, and flexors of the fingers, excepting the ulnar half of the deep flexor.

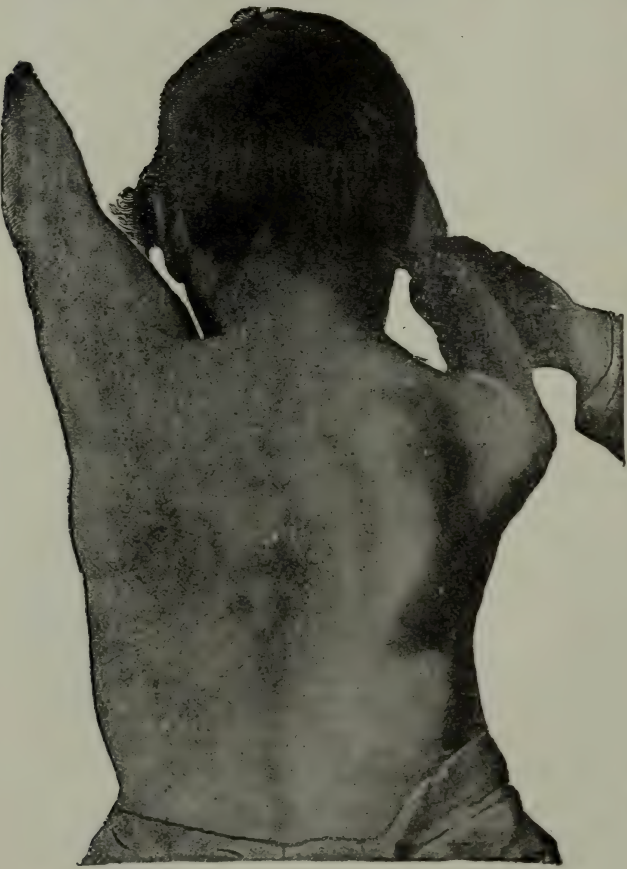

Fig. 158.—Paralysis of the Right Serratus. Winglike Protrusion of the Right Scapula When the Arm Is Stretched Forward. (After Strümpell.)

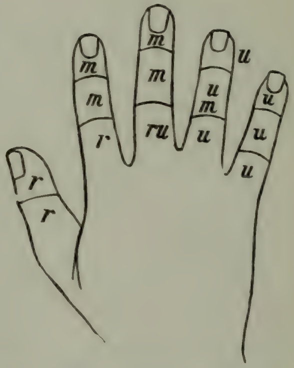

Fig. 159.—Detailed Distribution of the Nerves of the Dorsal Surface of the Fingers. (From Krause.) *r*, Radial Nerve; *m*, Median Nerve; *u*, Ulnar Nerve. (After Strümpell.)

If the lesion is high up, *paralysis* of all these muscles will occur, causing diminution of the power of flexing the wrist and pronating the forearm, inability to bring the thumb in contact with the ends of the fingers and to flex the second phalanges upon the first and in the index and middle fingers to flex the third.

If the lesion is just above the wrist, only loss of power in the thumb and fingers is present. The thumb muscles alone may be affected by the pressure on the thenar branch at the wrist. Atrophy and electrical changes (p. 610) in the affected muscles are usually present.

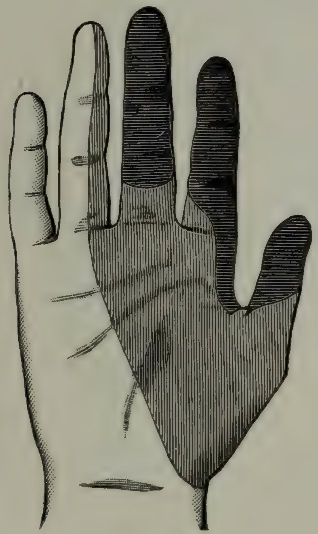

Fig. 160.

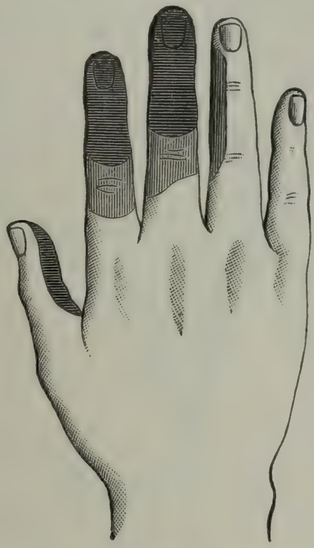

Fig. 161.

Figs. 160 and 161.—Showing Areas of Sensory Loss in Injuries of the Median Nerve. Horizontal Lines Show Total Anesthesia. Vertical Lines Show Partial Anesthesia. (After Bowlby; from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

Loss of sensation, if present, is shown in Figures 155, 159, 160 and 161. The nerve may be injured or divided by wounds or fractures of the forearm, at the wrist by pressure, or the seat of neuritis.

Volkmann's contracture may be mistaken for median nerve paralysis (p. 668).

7. *Ulnar Nerve*

The ulnar nerve arises from the inner cord of the brachial plexus and supplies the ulnar half of the flexor profundus digitorum, flexor carpi ulnaris, all muscles of the little finger, the interosseii, two ulnar lumbricalis, adductor pollicis, and inner head of the flexor brevis pollicis.

Paralysis due to a lesion high up causes impaired flexion of the hand on the forearm, inability to flex the first or extend the second and third phalanges, to oppose the thumb to the index finger, of abducting and adducting the fingers and to move the little finger. If the lesion is near the wrist paralysis of the intrinsic muscles of the hand, excepting those supplied by the median, occurs. *Sensation,* if lost, is in the areas shown in Figures 154, 155, 159, 162, and 163.

If the weakness persists for any length of time a characteristic deformity, due to the action of antagonistic muscles (p. 564), occurs. It is due to overextension and inclination of the hand to the radial side with hyperextension of the first phalanges (musculospiral) due to weakness of the flexors (ulnar) and flexion of the second and third phalanges (median) due to weakness of the extensors (ulnar). It is termed *main en griffe,* or claw-hand (Fig. 164). This appearance is still further accentuated by the atrophy of the intrinsic hand muscles supplied by the ulnar.

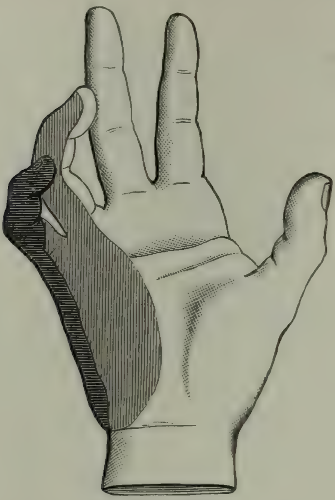

Fig. 162.

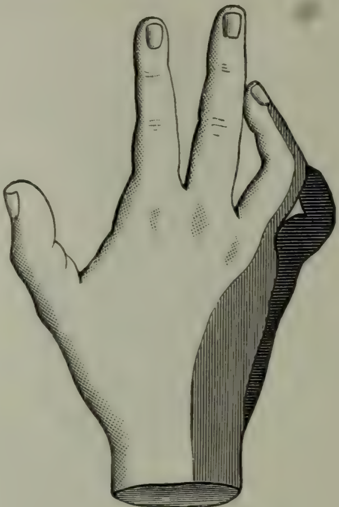

Fig. 163.

Figs. 162 and 163.—Showing Sensory Loss and Abnormal Position After Injuries of the Ulnar Nerve. (After Bowlby; from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

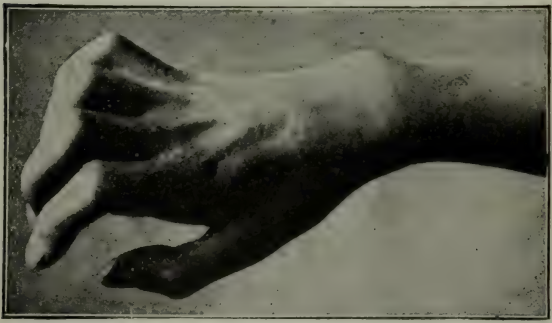

Fig. 164.—Paralysis of the Ulnar Nerve. Atrophy of the Interossei. The Terminal Phalanges Cannot Be Extended. (From the Erlangen Medical Clinic.)

In addition to neuritis, the nerve may be injured by pressure or traumatism at the elbow or wrist, fractures of the forearm, long-continued flexion of the elbow, cervical rib and stoop shoulders (p. 661).

Paralysis and atrophy of the intrinsic hand muscles (median and ulnar) are usually the first symptoms of progressive spinal muscular atrophy, amyotrophic lateral sclerosis, syringomyelia and cervical pachymeningitis.

8. *Combined Paralysis of Brachial Plexus*

Combined paralysis of the brachial plexus, in which all or nearly all the trunks are involved, may be due to: (1) dislocation of the head of the humerus, (2) fractures of bones of the arm, (3) ascending neuritis (p. 668), (4) injuries received during birth or later in life, (5) new growth in the neck, (6) primary brachial neuritis, (7) stoop shoulders (p. 667), (8) tearing of the roots from the spinal cord.

The **symptoms** vary according to the nerves affected and the severity of the lesion. The functions of each part of the plexus are described above. If mild in degree, sensory paralysis may not be present (p. 576).

Somewhat similar symptoms may be due to a lesion within the spinal canal in the cervical region, such as tumor, meningitis, or vertebral disease. When such is the case there would be evidences of irritation of the posterior roots, causing shooting pains down the arm, with absence of tenderness over the nerve trunks and possibly irritation of the anterior roots, causing spasm before paralysis occurred.

Types.—There are two types especially liable to occur: that of *Erb, or the upper arm type*, and that of *Klumpke, or the lower arm type.*

THE UPPER ARM TYPE usually is due to injury of the fifth and sixth cervical nerves (Fig. 153) at the side of the neck in front of the edge of the trapezius. In adults it may be due to pressure on the neck from carrying heavy weights on the shoulder, to neuritis, and it sometimes follows operations when the arms have hung in such a position as to put the nerves on the stretch (anesthetic paralysis). It is most commonly seen in young children, and in such has been termed *obstretrical paralysis, or the birth palsy of Duchenne.* The paralysis involves the deltoid, biceps, brachialis anticus, spinator longus and supra- and infraspinati. If other roots are affected (*infra*), other muscles may also be involved (Fig. 165).

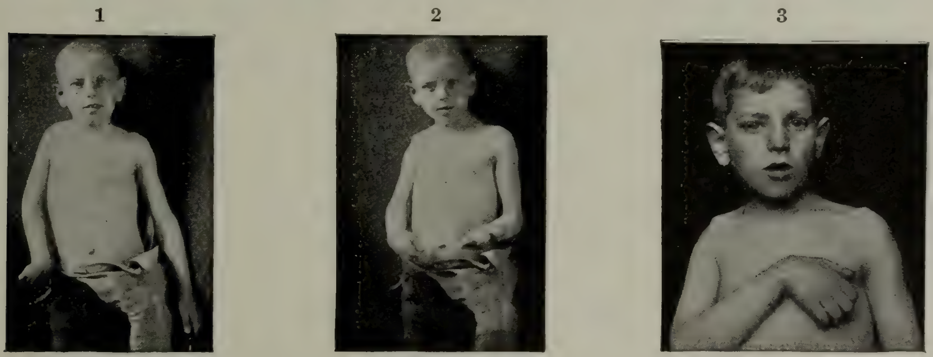

Fig. 165.

1. Typical Laceration in Brachial Birth Palsy. Ordinary Position in Which Patient Held Right Arm Before Operation.
2. Amount of Supination of the Right Hand Possible Before Operation.
3. Showing Extent of Muscle Power in the Biceps and Deltoid Before Operation.

(After Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

Atrophy of these muscles and changed electrical reactions (p. 610) are present. It is especially apt to occur after difficult delivery, as then there is apt to be excessive traction either on the head or shoulders and consequent stretching and laceration of the nerve trunks. In old cases there is lack of growth in the affected limb and inward rotation of the arm, pronation of the hand and a posterior subluxation of the humerus.

These cases must be distinguished from acute anterior poliomyelitis and paralysis due to cerebral palsy. It is frequently not discovered until the child is several months old, when it will be noticed that the affected arm is not used as the other.

The Lower Arm Type is due to involvement of the seventh and eighth cervical and first dorsal roots. The muscles affected are the triceps, pronators and flexors of the wrist, flexors and extensors of the fingers and the muscles of the hand. Ocular symptoms due to involvement of the cervical sympathetic are often present (p. 594).

The first dorsal root is sometimes alone affected, in which case the paralysis is limited to the muscles supplied by the median and ulnar nerves (*supra*), anesthesia in the ulnar distribution and eye symptoms above mentioned (Klumpke's paralysis).

The *symptoms* are weakness, atrophy and changed electrical reactions of the affected muscles. From lesions in the gray matter of the cord (poliomyelitis), the history, presence of tenderness over the nerve trunks, if a neuritis, or root pains, if due to an intravertebral lesion (p. 567), will usually suffice. Sensory paralysis is not present in poliomyelitis, but is not always present in disease of the nerves; its presence, however, will indicate the latter. Pain in the course of these nerves has been discussed on page 575.

(*b*) *Dorsal Nerves*

Symptoms of paralysis of these nerves are not discoverable. They are frequently the seat of pain (See p. 575).

Lumbar and Sacral Plexus

LUMBAR PLEXUS

The lumbar plexus is composed of the first three lumbar roots and one-half the fourth (Fig. 166). Its branches are the iliohypogastric and ilio-inguinal from the first lumbar, the genitocrural from the second lumbar, the external cutaneous from the second and third lumbar, and obturator and anterior crural nerves from the third and fourth lumbar roots.

It supplies sensory fibers to the skin of the lower part of the thigh, anterior portion of the scrotum (p. 627) in the male and labium in the female and testicle through the iliohypogastric and ilio-inguinal; the skin of the anterior and upper part of the thigh through the genitocrural; the skin of the anterior and upper portion of the thigh by the external cutaneous; the lower and anterior portion of the thigh by the middle

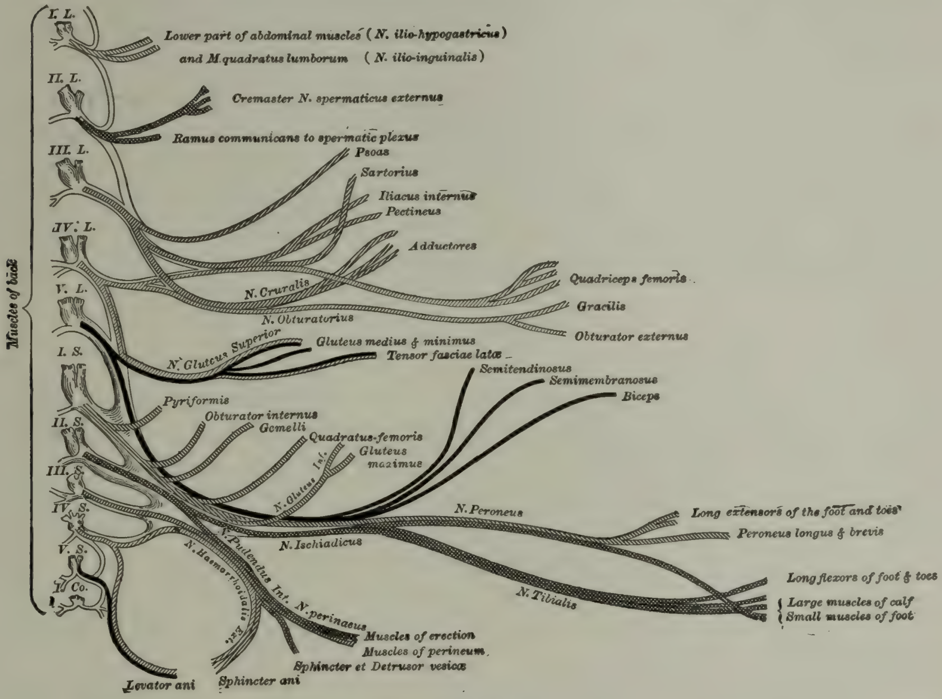

Fig. 166.—The Lumbosacral Plexus and Its Branches. (After Kocher; from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

cutaneous branch of the anterior crural, the inside of the thigh by the internal cutaneous branch of the anterior crural and the inside of leg between the knee and great toe by the long saphenous branch of the anterior crural (Figs. 156 and 156a).

1. *Obturator Nerve*

The obturator nerve supplies the adductors of the thigh; its paralysis causes loss of that function.

2. *Anterior Crural Nerve*

This nerve supplies the iliacus, pectineus, and all the muscles of the front of the thigh except the tensor vaginæ femoris.

Paralysis due to a lesion within the pelvis causes inability to flex the thigh on the body (iliacus) and to extend the leg on the thigh (quadriceps). If the lesion is outside the pelvis there is inability to extend the leg on the thigh. The knee jerk will be lost and other evidences of peripheral neuron lesions will be present. Loss of sensation may occur, as in Figure 156a.

The *nerve roots* may be damaged in the vertebral canal by disease of the vertebra (p. 733), spinal tumor (p. 734), meningitis (p. 683), and meningeal hemorrhage (p. 727). They are usually the earliest to suffer in tabes.

Within the abdomen the nerves may be pressed upon by abdominal tumors, psoas abscess, or a pregnant uterus. The obturator and anterior crural nerves have been injured during parturition, dislocation of the hip and wounds or blows in the groin. These branches may also be involved in disease of the cauda equina (p. 629) and suffer from inflammation due to any of the causes of multiple neuritis (p. 671).

Sacral Plexus

The sacral plexus is composed of the lumbosacral cord and the anterior divisions of the three upper and part of the fourth sacral nerves (Fig. 166). It supplies all the muscles of the leg excepting those supplied by the obturator and anterior crural, the sphincter of the bladder and anus and the genital organs (p. 592).

Within the spinal canal the roots form most of the cauda equina (pp. 624, 629); within the pelvis, the plexus may be damaged by tumors, pelvic inflammation, enlarged uterus from any cause, disease of the sacro-iliac joint (p. 666).

Paralysis of the small sciatic causes loss of power in the gluteus maximus, shown by difficulty in arising from the sitting position. There may be sensory loss in the perineum and middle third of the posterior part of the thigh and calf (Fig. 156).

3. Sciatic Nerve

The sciatic nerve supplies the muscles of the back of the thigh. Its *internal popliteal branch* supplies the popliteus, tibialis posticus, flexor longus digitorum, flexor longus pollicis and the muscles of the sole of the foot. The *external popliteal, or peroneal branch*, supplies the tibialis anticus, extensor longus digitorum, extensor brevis digitorum, extensor proprius hallucis, and peroneal muscles. The sensory distribution is shown in Figures 156 and 156a.

Paralysis, if due to a lesion near the sciatic notch, causes loss of power in the flexors, in the leg on the thigh, extensors of the thigh, and of all the muscles below the knee.

If the *internal popliteal* alone is involved there will be loss of the power of extending the foot upon the leg (plantar flexion), and of raising the body upon the toes. If the lesion is above the origin of the popliteus branch there is loss of inward rotation of the leg when flexed. Sensory loss, if present, is shown in *cpp,* Figure 156.

The *external plantar branch* supplies the muscles of the little toe, the flexor accessorius interosseii, the two outer lumbricals and the adductor of the big toe. Paralysis of these muscles causes a deformity similar to claw-hand (p. 660) and difficulty in walking. Sensory loss may be found in the small toe, adjacent half of the fourth and corresponding area of the foot. Paralysis of these muscles may be the first sign of beginning progressive neuritic atrophy.

The *internal plantar branch* supplies the short flexor of the toes, intrinsic muscles of the big toe except the adductor and the inner lumbricalis. The sensory supply is to the inner part of the sole, plantar surface of three inner toes and the adjacent half of the fourth.

If the *external popliteal* is alone involved, there is inability to flex the foot upon the leg (dorsal flexion), to evert the foot and to extend the toes on the foot. If the paralysis persists, talipes equinus or equinovarus is apt to occur, due to unapposed action of the flexor muscles. Sensory loss, if present, is shown in Figures 156 and 156a.

The popliteal nerves are often paralyzed by pressure against the edge of a chair. They are frequently first affected in multiple neuritis and progressive neuritic muscular atrophy, and rarely are affected early in progressive spinal muscular atrophy.

SCIATICA: The sciatic nerve is a frequent seat of pain, which is known as sciatica. The lesion is usually a perineuritis, most marked at the sciatic notch. It may extend to the interstitial tissue and eventually involve the nerve fibers. Some cases seem to be a true toxic neuralgia without demonstrable lesion (p. 573).

It is most common in middle life or after, in males than in females and in those whose general nutrition is poor. Those with a gouty diathesis are especially liable, and it frequently follows exposure to cold. Traumatism in the region of the buttock, and severe muscle strain are causes (See Neuritis).

Symptoms.—The symptoms are pain along the course of the nerve and its branches, although it may be confined to the region of the sciatic notch. This may be absent during rest, but is excited at once by walking. It may also be aggravated by sitting on a hard chair. Flexing the thigh on the body, thus stretching the nerve, usually aggravates the pain (Lasegue's sign). Constipation also aggravates it.

Tenderness is found along the course of the nerve, especially over the sciatic notch, and frequently in the popliteal space and below the head of the fibula. The patient is apt to stand or walk with the body leaning away from the affected side and the knee slightly flexed. The Achilles jerk is often absent. In severe cases motor paralysis and muscular atrophy may occur.

Differentiation.—The condition is usually unilateral, but bilateral involvement has occurred. Pain in this region from many other causes has been mistakenly called sciatica.

The conditions to be differentiated from sciatica are:

Intrapelvic disease, which can be discovered usually by proper examination and the absence of tenderness over the nerve.

Hip joint disease, which also can be discovered by examination of the joint.

Vertebral disease. In this there will be tenderness over the vertebra, and on jarring the body, possible deformity and the evidence of the skiagram.

Tabes dorsalis. See p. 753.

Disease of the cauda equina. See p. 629.

Disease of the sacro-iliac joint. This is comparatively common and may be either inflammation or relaxation due to either muscular strain or injury. It usually follows a fall or heavy lifting (sciatica may also be due to these causes).

X-ray examination may be sufficient. In addition it will be found that the patient gets up from a low chair with the back held stiffly; if he bends sideways from the hips there is limitation of motion on the affected side. Applying Kernig's test will cause pain in the joint, which will also be caused by grasping the iliac crests, separating them and then pushing them together.

Pain is apt to be pronounced while lying in bed and the patient will often place something hard under the back to support it. In true sciatica the pain is usually benefited by rest in bed.

Some cases of sciatic pain are due to arteriosclerosis (See p. 668). An enlarged transverse process of the fifth lumbar vertebra will cause it. This can be found by x-ray examination.

These nerves are injured in all spinal lesions below the first lumbar vertebra (cauda equina) and such may simulate extraspinal disease, especially sciatica. The lesion may be vertebral disease, vertebral injury (fracture, dislocation), hemorrhage, tumor, meningitis and neuritis. The first two are easily discovered by the existence of tenderness, deformity, and x-ray examination.

The symptoms due to the other causes are similar except that after hemorrhage the symptoms would appear acutely, and a history of traumatism is usually present.

In cauda equina lesions the pain is usually bilateral. Muscular paralysis of the peripheral neuron type (p. 563) gradually and progressively develops and the sphincters are apt to be involved. Sensation is usually more markedly interfered with than in extraspinal lesions (See also pp. 567, 629, 630).

3. Compression Palsy or Pressure Palsy

This is a paralysis due to long-continued pressure upon the nerve supplying the muscles and skin area affected. The pressure is usually exerted from the outside but it may be within the body, as brachial or ulnar paralysis due to cervical ribs or stoop shoulders which causes compression of the axillary structures between the humerus and the ribs, and the pressure of callus following a fracture.

Sensory symptoms are frequently absent (See p. 576) and if present consist of numbness, heaviness and tingling as when one's "foot is asleep." There may be slight loss of sensation at first, which usually soon disappears. The paralyzed muscles rarely atrophy and the reaction of degeneration is not usually present.

The *musculospiral* is most frequently affected owing to the habit of sleeping with the head resting on the arm. It is especially apt to occur after the patient has been drinking, and for this reason has been called "Saturday night paralysis." In such cases he awakens with the hand feeling numb and wrist drop (p. 657) present. The brachial plexus may be affected from using a crutch or if the arm hangs down and presses against the side of a bed or table as during operations (p. 661). The ulnar, sciatic, popliteals and anterior tibial are more or less frequently affected.

Differentation.—The condition must be distinguished from NEURITIS.

The history will usually make the diagnosis clear; in addition there is no pain or tenderness over the affected nerve. In neuritis due to lead, pain and tenderness are usually absent, and wrist drop is one of the earliest symptoms but it is bilateral and other symptoms of lead poisoning are present.

4. Neuritis

This may be confined to a single nerve and is termed *local,* or a number of nerves are affected, when it is termed multiple. It may be *interstitial,* when the inflammation is confined to the connective tissue, or *parenchymatous,* when the nerve fibers are primarily involved. If the perineurium is alone affected it is spoken of as *perineuritis.*

(*a*) *Local Neuritis*

Local neuritis may be due to exposure to cold (rheumatic or refrigeration palsy); traumatism, as blows over a nerve, wounds, tearing and stretching, which follow a dislocation or fracture, electrical shock; extension of inflammation from neighboring parts, as neuritis of the facial nerve due to inflammation of the middle ear, from septic wounds and inflammation of joints; arteriosclerosis; poisons (as multiple neuritis). It is usually of the interstitial variety.

Symptoms.—The symptoms consist of pain in the course of the nerve. This is apt to be burning and boring in type and worse at night; it is also increased by movement of the affected limb. Tenderness over the nerve is present; the skin may become thinned and glossy and the nails thickened and brittle; diminished sensibility or complete loss in the area supplied by the affected nerve (Figs. 154-156a) may or may not be present (p. 576). Function of the muscles supplied by the affected nerve is impaired; this may range from weakness to complete loss of power according to the severity of the lesion. More or less atrophy and change in the electrical reactions (p. 610) occurs in marked cases. Increased perspiration in the affected limb is sometimes noticed. The inflammation, especially when due to septic causes, may extend up the nerve, and involve others in the same plexus or even extend to the spinal cord; this is known as *ascending neuritis.* In mild cases the principal symptoms are pain and tenderness over the affected nerve.

Conditions to Be Differentiated from Local Neuritis

The condition must be distinguished from:

Pressure palsy
Division of a nerve
Neuralgia
Neuromata (p. 677)
Volkmann's contracture.
Flat feet.

PRESSURE PALSY.

The differential diagnosis is given on page 667.

DIVISION OF A NERVE.

Division of a nerve should be recognized by the history of an incised wound in the neighborhood of a nerve with complete motor and sensory paralysis in its distribution immediately following and continuing.

NEUROMATA.

Neuromata cause symptoms resembling neuritis. The diagnosis will depend upon finding the tumor or tumors by palpation over the nerve trunks.

NEURALGIA.

The differential diagnosis is given on page 574.

VOLKMANN'S CONTRACTURE.

Volkmann's contracture, or ischemic paralysis, is caused by the pressure of splints upon the forearm. The flexor muscles are affected; tenderness is not limited to the nerve trunks; sensation is not diminished or lost; and any muscle tissue left reacts normally to the electrical current.

Flat Feet.

Flat feet cause pain in the feet, extending up the leg. Examination will reveal this condition, if present.

(*b*) *Primary Brachial Neuritis*

Primary brachial neuritis is a primary inflammation of several or all of the nerves which compose the brachial plexus. It may be confined principally to the roots (radicular neuritis).

Occurrence.—It is not common and usually occurs in debilitated individuals past fifty years of age.

Symptoms.—The most prominent symptom is pain, which is usually very intense and most marked in the axilla and supraclavicular region, whence it may extend down the arm. It is aggravated by movement. There may be hyperesthesia and tenderness over the plexus. Loss of motor power often is not excessive, although it may be and accompanied by atrophy, glossy skin, vasomotor disturbances, and adhesions in the joints.

Conditions to Be Differentiated from Primary Brachial Neuritis

It must be distinguished from:

Neuralgia
Aortic aneurism
Cervical rib
Rheumatic arthritis
Vertebral disease
Cervical meningitis
Spinal tumor in cervical region
Cervical tabes.

Neuralgia.

From neuralgia, if the motor symptoms of neuritis are present, the distinction is easy (pp. 574, 668); when they are not, it is difficult. Most cases of brachial neuralgia—so-called—are due to some organic cause, all of which must be eliminated before a diagnosis of pure neuralgia can be made (p. 575). Tenderness over the nerve trunks, glossy skin and vasomotor symptoms indicate neuritis.

Aortic Aneurism.

Aortic aneurism may cause pain extending down the arm; tenderness will not be present and the physical signs of aneurism will be found.

Cervical Rib.

Cervical rib is found by x-ray examination.

RHEUMATOID ARTHRITIS.

Rheumatoid arthritis may be confounded when adhesions and partial ankylosis of a joint has occurred. In this condition, however, the pain is first localized in the affected joint with swelling and tenderness there, and other joints will sooner or later be affected. X-ray examination of the joints gives a characteristic picture.

VERTEBRAL DISEASE.

Vertebral disease will be discovered by the presence of tenderness over the affected part, pain when the body is jarred, possible kyphosis and the appearances found by x-ray examination.

CERVICAL MENINGITIS.

Meningitis, especially cervical pachymeningitis (p. 683), causes pain and motor paralysis in the arms. It is usually bilateral and attended with rigidity of the neck, pressure on the cord may cause weakness of the legs of the central neuron type (p. 563) and there is absence of tenderness over the nerve trunks.

SPINAL TUMOR.

Spinal tumor will sooner or later cause pressure on the cord with paralysis below the seat of the lesion. If the anterior roots are also involved muscular spasms may also occur. There will probably be sensory paralysis of the segmental type (p. 579).

CERVICAL TABES.

Tabes dorsalis affecting the cervical region causes shooting pains in the arms. Other symptoms of tabes will be found.

The nerve roots composing the *cauda equina* may also become inflamed and simulate tumor or other disease affecting that region. The diagnosis depends upon finding the condition at operation or improvement in the symptoms which will not occur if other causes are operative (pp. 629, 664).

(c) *Multiple Neuritis*

Multiple neuritis is practically always due to the influence of a poison of some sort.

Causes.—These are:

(1) *Idiopathic* (nature of poison unknown);

(2) *Poisons introduced from without;*

(a) Alcohol, carbon monoxid; carbon bisulphid; dinitro benzin; anilin. (b) Infectious diseases: diphtheria, influenza, typhoid fever, scarlatina, measles, etc., septicemia, tuberculosis, gonorrhea, syphilis, malaria, beriberi, leprosy. (c) Metallic poisons: lead, arsenic, mercury, phosphorus, silver, cyanid of potassium. (d) Ptomains.

(3) *Poisons produced within the body:* Gout, pregnancy and the puerperal state, diabetes.

(4) Dyscrasiae, chlorosis, marasmus, cancer and other forms of cachexia; arteriosclerosis.

Many of these as etiological factors are rare.

Symptoms.—In general it may be stated that the symptoms are characterized by pain and tenderness in the course of several nerves (diphtheria and lead are exceptions (*infra*)) and motor paralysis of the peripheral neuron type (p. 563). When occurring in children it may have to be distinguished from *poliomyelitis* (See p. 188) and in young infants pseudoparalysis due to scurvy may be thought to be multiple neuritis. Examination of the mouth and joints and the occurrence of hemorrhages will show the presence of scurvy.

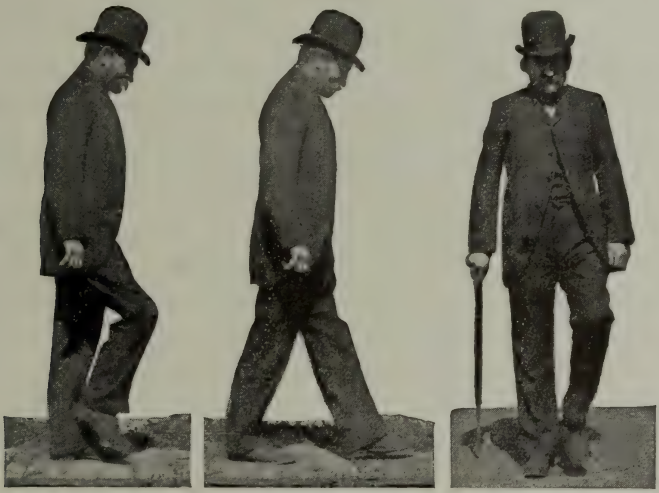

Fig. 167.—Gait in Multiple Neuritis, Showing the High Knee-action and the Dangling Foot Descending by Its Toe and Outer Border. (After Church and Peterson's "Nervous and Mental Diseases," published by W. B. Saunders Co., Philadelphia.)

Some of the above causes affect the sensory fibers principally, others the motor. Certain of them may cause symptoms peculiar to that cause. The more common of these merit separate consideration.

ALCOHOLIC MULTIPLE NEURITIS.

Alcoholic neuritis especially affects the sensory fibers and usually the first symptom complained of is numbness of the hands and feet, followed soon by pain, usually severe, in the course of the nerves and marked tenderness over them. Hyperesthesia of the calf muscles, elicited by squeezing them, appears early. Examination at this time will usually show absence of the knee jerks,[1] and weakness of the extensor muscles, those of the feet being usually the first affected. Double wrist drop and foot drop are characteristic symptoms. If the patient is able to walk the foot drop causes a peculiar gait, characterized by lifting the knees high in order that the toes may clear the ground. It is known as the *steppage gait* and is always present where there is weakness of the tibialis anticus and common extensor of the toes (Fig. 167).

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[1] During the first days they may be increased.

Other muscles, even those supplied by cranial nerves, may later become affected. The optic nerve is rarely the seat of neuritis. All the symptoms characteristic of peripheral neuron paralysis (p. 563) are present; sensory loss may or may not be. In some cases the motor fibers suffer slightly, and symptoms of sensory fiber involvement predominate. In such cases muscular incoördination may be present (acro-ataxia, see p. 587), this condition being known as *pseudotabes.*

Mental symptoms are frequent and characterized by mental confusion. There is more or less loss of memory, hallucinations—especially of sight and hearing, a tendency to tell at length various imaginary happenings (confabulation) and disorientation. This condition is known as *Korsakow's disease,* or *polyneuritic psychosis.*

In severe cases there may be elevation of temperature, weakness of the heart, and death.

Differentiation.—It must be differentiated from TABES DORSALIS (p. 756), ACUTE POLIOMYELITIS (p. 188), TRANSVERSE MYELITIS (p. 731) and MULTIPLE MYOSITIS. In this the tenderness is confined to the muscles; the nerves are not tender. Reactions of degeneration are not present and the apparent weakness is due largely to the pain caused by motion. It must be borne in mind that steady and often secret drinking is more likely to cause the condition than the going on periodical sprees and that women are more liable than men. (See also p. 675.)

NEURITIS DUE TO LEAD.—Neuritis due to lead is characterized by the fact that the sensory nerve fibers are but slightly or not at all affected; hence pain and tenderness are rarely present. The posterior interosseous nerves are usually first affected, causing bilateral wrist drop (p. 657). It is usually the case that the supinator longus escapes and stands out prominently in contrast with the other muscles which are atrophied. The *opponens pollicis* may also escape. Other nerves may be affected, and the usual signs of lead poisoning are present (*vide*). Lead may also cause degeneration of the anterior horn cells (See p. 194).

DIPHTHERITIC NEURITIS.—Diphtheritic neuritis is also characterized by the usual escape of sensory nerve fibers and the absence of pain and tenderness over the affected nerves. Cranial nerves are frequently first affected, and nasal voice due to paralysis of the muscles of the palate is usually the first symptom. This is followed by difficulty in swallowing, shown by the regurgitation of liquids. Inspection will show the palate to hang lower than normal and the palatal reflex will be absent. The muscles of the eye are next affected or may be the first to suffer. This is usually an ophthalmoplegia interna (p. 635). The muscles of the limbs may or may not be involved. The pneumogastric nerve is especially liable, as shown by rapid, irregular and weak heart. The symptoms usually appear during the convalescing period. Loss of the knee jerk occurs very early.

Diphtheria may also cause either inflammation of the cortical cells and cranial nerve nuclei (p. 715), or paralysis due to cerebral apoplexy (p. 689). When due to the latter the paralysis is hemiplegic and of the central neuron type.

ARSENICAL NEURITIS.—Arsenical neuritis is characterized by pain and numbness of the limbs and tenderness over the nerve trunks, followed by paralysis, especially of the extensors. It may be due to medicinal doses of arsenic long continued. Other evidences of chronic arsenical poisoning are apt to be present (*vide*).

SENILE NEURITIS.—Senile neuritis is due to arteriosclerosis and consequent diminution of the supply of blood to the nerves. The symptoms are numbness of the hands and feet, and weakness with loss of reflexes and slight muscular atrophy. There is usually some tenderness. Cranial nerves may also be affected.

BERIBERI.—Beriberi has been described on page 230.

IDIOPATHIC NEURITIS.—Idiopathic neuritis depends for its diagnosis upon the presence of the symptoms of neuritis and the inability to find any other adequate cause.

RECURRENT NEURITIS.—One attack seems to predispose the patient to others, no matter what the cause. This is especially true of that due to lead. Cases, however, have been described in which there were a number of recurrences without known cause.

5. Progressive Neurotic Muscular Atrophy

Occurrence.—This disease, also known as neurotic muscular atrophy, peroneal type of progressive muscular atrophy, Charcot-Marie-Tooth disease, and neural progressive muscular atrophy of Hoffmann, occurs usually in early life, before the twentieth year, but may appear later. It is a hereditary or family disease, but sporadic cases occur.

Symptoms.—The lesions found are degeneration of the peripheral nerve fibers, associated with more or less degeneration in various regions of the cord, especially the columns of Burdach.

The symptoms consist of atrophy and weakness which first appear in the small muscles of the feet and soon spread to the tibialis anticus, extensor longus digitorum, extensor hallucis longus and peroneal muscles. This weakness causes deformity of the feet, at first resembling "claw-hand" (p. 660), followed by the development of pes cavus, then pes equinus or equinovarus, and a steppage gait (p. 672) (Fig. 167). As the calf muscles become affected this may disappear and the appearance shown in Figure 168 result. Sooner or later the small muscles of the hand and those of the forearm become involved, as in spinal progressive muscular atrophy (Fig. 175). Fibrillary twitchings are usually seen in the affected muscles, the tendon reflexes are diminished or lost and various degrees of DeR are found. Vague dull pains and paresthesias in the limbs are usually complained of and diminished sensibility may be found in some cases. The affected limbs are cyanotic and cold. Rarely do the symptoms begin in the hands. Cases have been described in which, associated with these symptoms, there were also optic atrophy, mental impairment, and paralysis of cranial nerves, and others have been described in which cataract occurred.

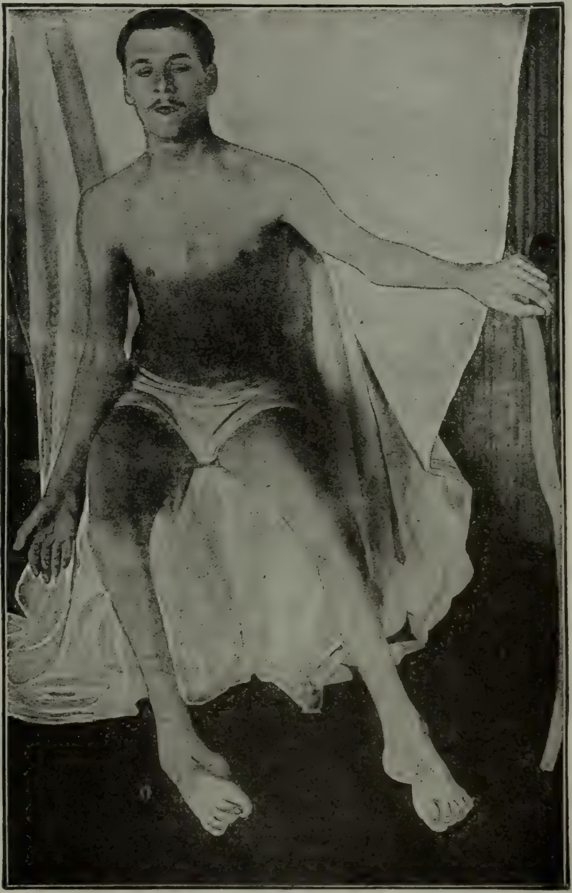

Fig. 168.—Neurotic Muscular Atrophy. The Disease Came on in Almost the Same Fashion in Three Members of One Family. (After Strümpell.)

Conditions to Be Differentiated from Neuritic Progressive Muscular Atrophy

The disease must be distinguished from:

Neuritis
Progressive spinal muscular atrophy
Muscular dystrophy
Acute poliomyelitis
Interstitial neuritis
Friedreich's ataxia.

NEURITIS.

Neuritis is distinguished by pain and tenderness over the nerve trunks (except as mentioned on p. 671). The symptoms follow one of the causes of multiple neuritis, and they develop more quickly.

	Neuritic Atrophy	Neuritis	Progressive Spinal Atrophy	Dystrophy	Interstitial Hypertrophic Neuritis
Onset	Gradual. Early life	More rapid but not necessarily acute. Any age	Gradual. After thirty	Gradual. Early life	Gradual. Early life
Muscles first attacked	Small muscles of feet followed by those of leg	Usually extensors of hands or feet. Muscles of forearm or leg	Small muscles of hands usually. Peroneal group or others rarely	Shoulder girdle, or muscles of face	Muscles of feet
Fibrillary tremor	Often present	Seldom present	Always present	Never present	Often present
Electrical changes	More or less marked; either quantitative decrease or De R	Same	May be normal at first, followed by quantitative decrease. De R may occur late	Normal or quantitative decrease	Same as neuritic atrophy
Sensory symptoms	Slight pain or paresthesia	Usual severe pain	No pain	o pain	Same as neuritic atrophy
Palpation of nerves	Negative	Tender	Negative	Negative	Felt as thickened cords. (Do not confuse with neuromata (p. 677), which are isolated tumors)
Course of disease	Slowly progressive	At first progressive, followed by improvement and recovery	Slowly progressive	Slowly progressive	Slowly progressive

Progressive Spinal Muscular Atrophy.

Progressive spinal muscular atrophy occurs usually later in life (past thirty). The atrophy begins in the small muscles of the hand, and the muscles of the back, shoulders and chest sooner or later become affected; sensory symptoms are absent; it is not hereditary. If the symptoms begin in the muscles of the feet and legs and early in life, as they may sometimes do, the distinction at first may be difficult.

Muscular Dystrophy.

Muscular dystrophies do not have sensory symptoms and fibrillary tremors while the atrophy begins in the muscles of the shoulder girdle followed by involvement of those of the thigh. Pseudohypertrophy may also occur. It must be borne in mind that cases occur in which the two conditions may more or less overlap.

Acute Poliomyelitis.

Acute poliomyelitis is a disease of acute onset followed by more or less general paralysis with later retrogression. The history should be sufficient.

Interstitial Neuritis.

Hypertrophic interstitial neuritis is a very rare disease in which the symptoms resemble those above described but in which the thickened nerve trunks are palpable and incoördination occurs.

Friedreich's Ataxia.

The deformity of the foot in neuritic atrophy may be simulated by that occurring in Friedreich's ataxia (p. 700). In this, however, there are speech disturbance, nystagmus, usually the Babinski reflex, and ataxia.

6. Arthritic Muscular Atrophy

This is an intense atrophy and weakness occurring in the muscles about a diseased joint.

Causes.—It is probably due to a reflex disturbance caused by irritation of the articular nerve endings which is referred to the anterior horn cells and muscular nerve endings.

Symptoms.—Inflammation of a joint due to any cause may produce the symptoms which consist of atrophy, usually in the extensor muscles on the proximal side of the joint, often with fibrillary tremors and electrical reactions that are either normal, or show a quantitative decrease. The mechanical irritability of the muscles is increased. The atrophy and weakness are greater than can be accounted for by disuse.

Diagnosis.—The diagnosis depends upon the occurrence of the symptoms secondary to joint disease. It must be remembered that trophic changes in a joint causing more or less ankylosis may follow neuritis. In this, however, weakness, atrophy and pain in the course of the nerves precedes the development of joint trouble.

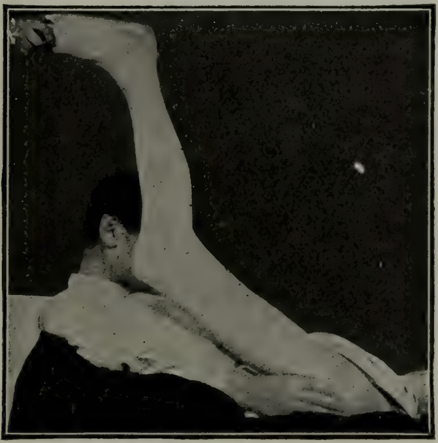

Fig. 169.—Hypotonia in Late Tabes dorsalis. (After Dana's "Textbook of Nervous Diseases," published by William Wood & Co., New York.)

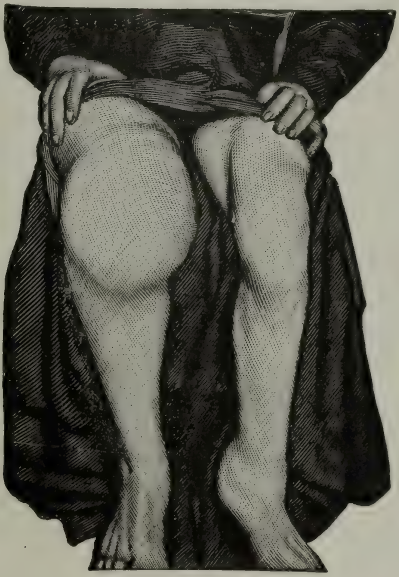

Fig. 170.—Tabetic Arthropathy of the Right Knee and Left Ankle. (After Strümpell.)

7. Neuromata or Tumors of Nerves

These may be of the nature of tumors elsewhere (pseudoneuromata), or consist of nerve tissue. They may be single or multiple and are frequently due either to traumatism or occur on the ends of nerves after amputation. They also may develop spontaneously and are apt to occur in myxedematous, cretinoid, and tubercular conditions. Pseudoneuromata are the more common. They are present in Von Recklinghausen's disease, in which molluscous tumors of different sizes are scattered over the surface of the body. Heredity seems to play a part.

Symptoms.—The symptoms resemble those of neuritis, viz., pain, tenderness, and weakness of the muscles supplied by the affected nerves.

Diagnosis.—The diagnosis depends upon finding the tumors by palpation.

E. Diseases of the Meninges

The most common of these, i. e., the epidemic cerebrospinal and tubercular meningitis, have been described elsewhere (pp. 52, 89). There are other forms of especial neurological significance. The inflammation may be confined to the dura (*pachymeningitis*) or to the pia arachnoid (*leptomeningitis*).

1. Cerebral Meningitis

(*a*) *Cerebral Pachymeningitis*

External Pachymeningitis.—External pachymeningitis may be due to traumatism to the head, sunstroke, caries of the cranial bones due to syphilis, middle ear disease, ozena, and secondary infection from inflammation of the skin of the face or head, as erysipelas.

Symptoms.—The symptoms are usually indefinite, consisting of severe headache, vertigo, slight mental confusion, and if of severe and acute type, fever, delirium and convulsions.

Diagnosis.—Examination of the cerebrospinal fluid will be negative, unless the pia arachnoid is also involved. The diagnosis depends upon the occurrence of the symptoms following one of the causes mentioned. It is often of a chronic type.

Internal Cerebral Pachymeningitis.—Internal cerebral pachymeningitis is met with in two forms, the purulent and the hemorrhagic. The former occurs usually in connection with a similar condition of the pia arachnoid, but may exist independently, as in a case described by Leszynsky, in which it was due to purulent disease of the middle ear. The latter is known as *hemorrhagic internal pachymeningitis.* It is usually found in old people who have been alcoholic or syphilitic, and in the chronic insane. It also occurs in cases of anemia from various causes. Ill-nourished and rachitic babies may have it.

Symptoms.—The symptoms of the purulent type are those of inflammation of the pia arachnoid (*infra*). Those of the hemorrhagic form are very indefinite and the condition is often found at autopsy without a diagnosis having been made.

Diagnosis.—The diagnosis may be tentatively made, if apoplectic attacks in which the symptoms are of brief duration (See also p. 713) occur in those who would be likely to develop the condition. At these times there may be slowness of the pulse, contracted pupils with poor response to light, vomiting, coma and hemiplegia more or less marked.

(*b*) *Cerebral Leptomeningitis*

Acute Cerebroleptomeningitis

This condition may be due to infection by either the pneumococcus, streptococcus, staphylococcus, typhoid bacillus, gonococcus, colon bacillus, bacillus of influenza, or those of other infectious diseases. The first two are probably the most common. It, therefore, may occur as a sequel of pyemia, septicemia or any of the infectious diseases. The organism of pneumonia may cause it without the coexistence of that disease. Extension from purulent disease of the frontal sinuses and upper nasal passages, and *middle ear,* are frequent causes. Spinal meningitis may coexist.

Symptoms.—The symptoms are similar to those referable to the cerebrum in cerebrospinal meningitis (p. 52). If due to purulent infection, high fever and chills occur.

Diagnosis.—The cause is discovered by its following or occurring during the course of one of the causes mentioned above and by examination of the cerebrospinal fluid, which will show the characteristics peculiar to the particular kind of meningitis existing, and also contain the peculiar organism causing it. It may be said that the fluid, except in the tubercular, resembles in characteristics that of the cerebrospinal.

The course of tubercular meningitis is usually not so acute (See Serous Meningitis, p. 680).

Conditions to Be Differentiated from Meningitis

In addition meningitis must be distinguished from:

Cerebral rheumatism
Cerebral syphilis
Meningismus;
Acute delirium.

CEREBRAL RHEUMATISM.

Cerebral rheumatism occurs in the course of acute articular rheumatism. Delirium, hallucinations, headache, and rigidity may then occur. The cerebrospinal fluid will be negative.

CEREBRAL SYPHILIS.

In cerebral syphilis the symptoms are usually not so acute. The cerebrospinal fluid is not purulent and responds to the tests for syphilis (p. 758).

MENINGISMUS.

Meningismus is a condition simulating meningitis, occurring in the course of the infectious fevers, especially typhoid and pneumonia. In it the cerebrospinal fluid is normal.

ACUTE DELIRIUM.

In acute delirium, or typhomania, cranial nerve palsies, rigidity, hyperesthesia, Kernig's sign, and convulsions do not occur. The cerebrospinal fluid is clear.

The *glycyltryptophan test* has been advocated to distinguish true meningitis from any of the above. This is made by placing 1 c.c. of the cerebrospinal fluid in a test tube with an equal quantity of glycyltryptophan;

then 1 c.c. of tolulol is added. The mixture is placed in an incubator for three hours after which a few drops of dilute acetic acid are added. An oversaturated solution of calcium chlorid is then added drop by drop, and a red color is produced if meningitis is present.

Epidemic Cerebrospinal Meningitis

This condition has been described on page 52.

Tubercular Meningitis

This condition has been described on page 89.

Chronic Cerebral Leptomeningitis

Chronic cerebral leptomeningitis may occur secondary to an acute attack or be primarily due to syphilis, alcoholism, traumatism, sunstroke. That due to syphilis is the most common (See pp. 747, 749). A basilar form may occur, especially in the cerebellar region, which stimulates tumor there. It is a cause of hydrocephalus.

Symptoms.—The symptoms are vague, consisting of headache, vertigo, convulsions of either the Jacksonian or general type, if the lesion is vertical; paralysis of cranial nerves and symptoms of hydrocephalus, if basilar. Optic neuritis or mild papilledema may occur.

Diagnosis.—The diagnosis, excepting the syphilitic form, can rarely be made with certainty but may be surmised if the symptoms follow the causes mentioned.

2. Serous Meningitis

(*Meningitis serosa*)

This consists of an abnormally large amount of cerebrospinal fluid existing under pressure, which causes symptoms resembling those of meningitis.

Causes.—Quincke believed that the exudation was analogous to a pleural effusion and was due to a vasomotor disturbance. It may be due to any of the infections causing acute cerebral meningitis (p. 678), and may occur during gastro-enteritis in young babies. When due to infections it is believed that the organisms are of slight virulence and not numerous enough to cause the formation of pus. Alcoholism is rather a frequent cause. There is an internal type in which the fluid is confined to the ventricles (acute hydrocephalus), and an external type in which it is in the pia. The former is common in infants with gastro-intestinal disturbances.

Symptoms.—The symptoms resemble those of meningitis of the ordinary type, but are usually not so acute in development and cause. Optic neuritis and papilledema are very common.

Alcoholic Form.—The alcoholic form, known also as postdelirious alcoholic stupor and alcoholic cerebral edema, follows frequently delirium tremens or a prolonged debauch. There is immobility of the facies, contracted pupils, rigidity of the neck, hyperesthesia and a muttering delirium.

Conditions to Be Differentiated from Serous Meningitis

The diagnosis of the alcoholic form depends upon a history of alcoholism; that due to other causes must be distinguished from:

Brain tumor
Uremia
True meningitis.

Brain Tumor.

In the absence of definite focal symptoms the diagnosis from brain tumor may be difficult. It may depend on the fact that in time the patient recovers either temporarily or permanently, and some cases of so-called pseudotumor are evidently of this nature. Lumbar puncture will cause marked relief. If the symptoms follow any of the causes mentioned it would be significant.

Uremia.

Uremia will be recognized by examination of the urine and previous history.

True Meningitis.

True meningitis is excluded by examination of the cerebrospinal fluid which in serous meningitis is expelled with great force, is clear, and may be sterile.

3. Hydrocephalus

Hydrocephalus, by which is meant an excess of cerebrospinal fluid, exists in two forms, external and internal. In the former the fluid is in the subarachnoid space and has been described under serous meningitis. In the latter, which is the more common, the fluid is in the ventricles. The external form may coexist with this. It may be either congenital or idiopathic, acquired or secondary.

Congenital or Idiopathic Form.—The etiology of this form is not understood. Alcoholism or syphilis in the parents seems to have etiological significance. It is frequently associated with other deformities, as spina bifida, harelip, cleft palate, etc.

Symptoms.—The symptoms appear very early in life, in fact they may begin *in utero.* They consist of gradual enlargement of the head, which becomes pear-shaped, the face retaining its usual size and the forehead bulging over the eye, the bones become abnormally thin, there are bulging of the fontanels, restlessness, irritability, poor nutrition, and, if the child lives, failure to develop mentally and physically with spastic paralysis of the limbs, optic atrophy, and usually epileptiform convulsions. In some cases the condition is arrested and the patient goes through life with the peculiarly shaped and enlarged head, more or less spastic paraplegia and lack of intelligence.

Acquired Form.—The acquired form may occur at any age and is due to anything that blocks up the passages between the ventricles and subarachnoid space (foramen of Magendie, foramina of Monro). It may be caused by basal meningitis due to any cause, tumor in the posterior fossa which makes pressure on the veins of Galen or aqueduct of Sylvius (cerebellar growths or those in the third ventricle usually), inflammation of the ependyma, and be part of a serous meningitis (p. 680). If any of these conditions occur before the fontanels have closed and sutures united, the symptoms are similar to those detailed above, but are preceded by those of the primary condition.

If they occur after the fontanels have closed and the sutures have united, enlargement of the head does not occur, although thinning of the bones may follow. Spastic paralysis and increased reflexes remaining after an attack of meningitis would be significant. When due to brain tumor occurring late in life the diagnosis is very difficult, in fact may be impossible. The rapid development of spastic para- or diplegia and increased reflexes occurring in the course of the symptoms of cerebellar or other subtentorial tremor are suspicious. The occurrence of a "cracked pot" percussion note when the skull is percussed is said to indicate its existence. A skiagram may also aid, as it will show the areas of bone atrophy if they are present. The fluid will not flow freely after lumbar puncture, as the communication between the ventricles and subarachnoid space is closed. It must be borne in mind, however, that lumbar puncture in subtentorial tremors may be dangerous.

Conditions to Be Differentiated from Hydrocephalus

When occurring in infancy or early childhood it must be distinguished from:

Rachitis
Spastic paralysis due to cerebral lesion or agenesia
Syphilitic thickening of cranial bones.

Rachitis.

In rachitis, the head while usually enlarged is square in shape, the bones are not thinned and the fontanels do not bulge, and the characteristic nervous phenomena are absent.

Spastic Paralysis.

Children with spastic paralysis due to meningeal hemorrhage at birth or to lack of cortical development (pp. 686, 689) will not have the peculiarly shaped head.

Syphilitic Thickening of Cranial Bones.

Syphilitic thickening of the cranial bones can readily be told by palpation and x-ray examination.

4. Spinal Pachymeningitis

Causes.—Spinal pachymeningitis may be due to vertebral caries or syphilis. There is, moreover, a special type which usually affects the cervical region, that is known as *pachymeningitis cervicalis hypertrophica of Charcot and Joffroy.* Rarely the lumbar region is similarly affected. Alcohol, syphilis, traumatism and exposure have been causes.

Symptoms.—The symptoms at first are shooting pains radiating from the neck down the arms; with this may be associated muscular twitchings or spasm. Atrophy, affecting first the thenar and hypothenar eminences and interosseous spaces (median and ulnar nerves, pp. 658, 659), soon follows. Other muscles of the arms become affected later. The reflexes are absent and fibrillary tremors are present.

As the disease progresses, pressure is made on the cord, paralysis of the legs of a spastic or central neuron type (p. 563) develops; the sphincters may or may not be intact; either dissociation of sensation (p. 577) or hypesthesia or hypalgesia may be present in the hands and arms. Symptoms of involvement of the cervical sympathetic (p. 594) are frequently present and the neck is apt to be rigid. If the disease is in the lumbar region similar symptoms occur in the legs, although if all the lumbosacral segments are involved the paralysis will be atrophic and flaccid (p. 630).

Conditions to Be Differentiated from Spinal Pachymeningitis

The diagnosis must be made from:

Syringomyelia
Amyotrophic lateral sclerosis
Progressive spinal muscular atrophy
Vertebral caries
Spinal tumor
Primary brachial neuritis.

SYRINGOMYELIA.

From syringomyelia, if dissociation of sensation is present, differentiation may be difficult. In it, pain, if present at all, is not great; there is no rigidity of the neck, and in meningitis the great thickening of the meninges may be shown by the x-ray.

AMYOTROPHIC LATERAL SCLEROSIS—PROGRESSIVE SPINAL MUSCULAR ATROPHY.

In amyotrophic lateral sclerosis there are no sensory symptoms; the same may be said of progressive spinal muscular atrophy.

VERTEBRAL CARIES.

Vertebral caries can be detected by the presence of kyphosis, pain on pressure, jarring, and x-ray examination.

SPINAL TUMOR.

Spinal tumor may be difficult; the symptoms, however, are more apt to be unilateral at first, and there would be no rigidity of the neck. Pachymeningitis occurs usually late in life, tumor at any age.

PRIMARY BRACHIAL NEURITIS.

Primary brachial neuritis does not give cord symptoms; muscular atrophy is not so apt to occur; the pain is constant.

Differentiation of Lumbar Type

The lumbar type may be mistaken, in addition, for:

Tabes dorsalis
Tumor of the cauda equina.

TABES DORSALIS.

In tabes dorsalis extensive muscular atrophy and paralysis are usually absent and the Argyll-Robertson pupil and other ocular symptoms usually present.

TUMOR OF THE CAUDA EQUINA.

It is usually impossible to distinguish tumor affecting the cauda equina from meningitis (See also pp. 629, 666).

5. Spinal Leptomeningitis

Spinal leptomeningitis may be either *acute* or *chronic*.

ACUTE FORM.—The acute form usually occurs associated with acute cerebral meningitis, especially the epidemic form (pp. 52, 678). It may also be traumatic and be associated with myelitis.

Symptoms.—If not associated with the cerebral form the symptoms would be pain in the course of the nerve roots, hyperesthesia, moderate opisthotonos, muscular irritability, and Kernig's sign, followed by paralysis, hypesthesia and hypalgesia.

Diagnosis.—The diagnosis depends on the same principles as govern that of cerebral meningitis (p. 679).

CHRONIC FORM.—The chronic type is usually associated with tubercular or syphilitic disease of the vertebra. It may follow traumatism or be associated with myelitis (p. 731).

Symptoms.—The symptoms are those of nerve root irritation and more or less weakness of the limbs below the seat of the lesion (See pp. 567, 683).

6. Serous Spinal Meningitis

A condition has been described by Horsley and others in which when the spinal canal was opened a large excess of fluid was found. This occurs in supposed cases of myelitis, disease of the vertebra, and syphilitic and tuberculous spinal meningitis. Also, a number of cases supposed to have spinal tumor have been found at operation to have a circumscribed collection of fluid in the pia arachnoid. This has been termed *circumscribed serous spinal meningitis.* Some cases have been believed to follow influenza, others trauma to the back.

Symptoms.—The symptoms are those of tumor (p. 736).

F. System Diseases

Certain diseases consist of degeneration or inflammation, confined either entirely or mostly to one or more of the tracts which have been described on pages 555, 556. These have been called system diseases.

1. Diseases of the Sensory Tracts

Posterior Poliomyelitis

Posterior poliomyelitis, also known as *herpes, herpes zoster,* and *zona,* is an inflammation of the ganglia, situated upon the posterior roots of the spinal nerves and those situated upon the sensory cranial nerves. It is most frequently found in the dorsal nerves and then causes the condition known as *herpes zoster,* or *shingles.*

Causes and Symptoms.—It is due to an infection.

The symptoms consist of neuralgic pain in the course of the affected nerves followed by an eruption of herpes in this area; there may be impaired sensation.

At first it is usually thought to be a simple neuralgia of the intercostal or fifth nerves (those usually affected), the diagnosis not being made until the eruption appears (See also p. 575).

Herpes may also be due to degeneration of these ganglion, as in tabes dorsalis.

2. Diseases of the Motor Neurons

These may affect either the upper or lower, or both.

(*a*) *Diseases of the Upper Motor Neurons*

1. Primary Lateral Sclerosis

This is a degeneration confined to the pyramidal tracts. It is rarely primary, but usually occurs as a descending degeneration due to cutting off the fibers from the parent cortical cells by a lesion, such as a localized myelitis or area of sclerosis, as found in multiple sclerosis.

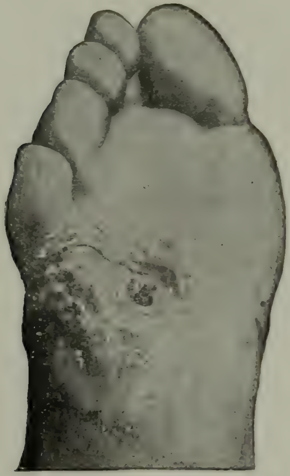

Fig. 171.—Perforating Ulcer of Foot in Locomotor Ataxia. (After Obersteiner; from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

It is most common between the ages of twenty and forty and seems to be due to either syphilitic infection, exposure, infectious diseases, or traumatism to the back.

A *hereditary form* has been described in which the symptoms appear either in childhood or young adults. In such cases it is due to an inherent lack of vitality and hence early death of the nerve fibers (abiotrophy) (See also pp. 686, 688, 694, 697, 698, 699).

Symptoms.—The symptoms consist of a gradually developing weakness of the legs, associated with great spasticity, increase of the tendon reflexes, and presence of the Babinski reflex. One leg may be affected some time before the other.

In marked cases the legs are held stiffly and close together and seem to be pushed forward, the feet sliding over the ground (Fig. 172). In some cases either talipes equinus or equinovarus may develop; in such the patient walks more or less on the ball of his foot. The sphincters may either not be affected until late in the disease or not at all, and sensory symptoms are absent.

Conditions to Be Distinguished from Primary Lateral Sclerosis

It must be distinguished from:

Transverse myelitis in the dorsal region
Posterolateral sclerosis

Hereditary amaurotic ataxic paraplegia
Atypical forms of multiple sclerosis
Amyotrophic lateral sclerosis
Lenticular degeneration
Secondary degeneration following apoplexy
Hysterical paraplegia.

Transverse Myelitis.

In transverse myelitis there will be loss of or diminished sensibility below the seat of the lesion. The sphincters are usually involved (p. 731).

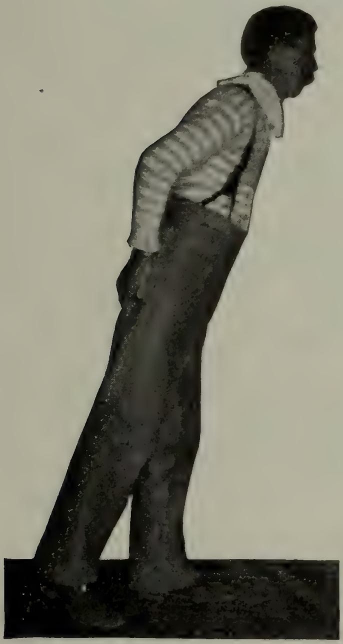

Fig. 172.

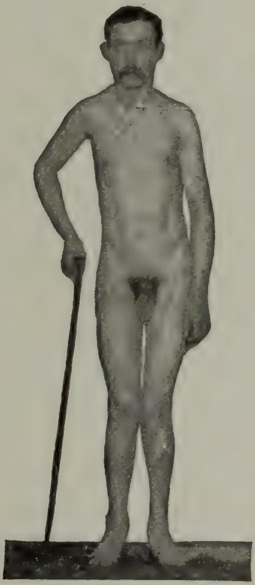

Fig. 172a.

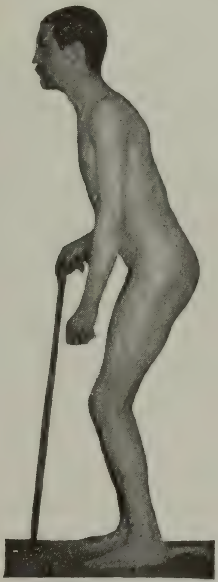

Fig. 172b.

Fig. 172.—Paraplegic Gait (After Church and Peterson's "Nervous and Mental Diseases," published by W. B. Saunders Co., Philadelphia.)

Fig. 172a and 172b.—Station in Spastic Paraplegia Due to Syphilitic Myelitis, Showing Rigidities, Flexed Knees, and Adducted Thighs. (After Church and Peterson's "Nervous and Mental Diseases," published by W. B. Saunders Co., Philadelphia.)

If the meninges are involved (meningomyelitis) there will be a girdle sensation.

Posterolateral Sclerosis.

The gait in posterolateral sclerosis is a mixture of the spastic and ataxic, and the Romberg symptom is present. In *subacute combined sclerosis* (p. 702) there is paresthesia and pain.

HEREDITARY AMAUROTIC ATAXIC PARAPLEGIA.

This condition may be mistaken for the hereditary form of lateral sclerosis. In the former there is atrophy of the optic nerve and the gait resembles that of posterolateral sclerosis (See p. 698).

ATYPICAL FORMS OF MULTIPLE SCLEROSIS.

Atypical forms of multiple sclerosis, in which the diseased areas involve principally the pyramidal tracts, may be difficult to distinguish. Examination will usually show pallor of the temporal halves of the optic disk and slight intention tremor.

AMYOTROPHIC LATERAL SCLEROSIS.

In amyotrophic lateral sclerosis, in addition to the spasticity of the lower limbs, there will be muscular atrophy, especially in the muscles of the hand.

LENTICULAR DEGENERATION.

The points of difference will be noticed if the description of this disease is read (*infra*).

SECONDARY DEGENERATION FOLLOWING APOPLEXY.

The history of one or more attacks of apoplexy makes plain the origin of the spastic gait due to secondary degeneration.

HYSTERICAL PARAPLEGIA.

In hysterical paraplegia the Babinski reflex will be absent; a true ankle clonus is exceedingly rare. Other stigmata of hysteria will usually be found if looked for (p. 775) and the paralysis is apt to develop suddenly after a mental or physical shock.

2. Unilateral Progressive Ascending and Unilateral Progressive Descending Paralysis

Symptoms.—The symptoms begin in the lower limb and gradually ascend, or vice versa. Such cases are rare.

According to Mills, who first described them, they may be caused by (1) primary degeneration of the pyramidal tracts; (2) as an early stage of multiple sclerosis; (3) unilateral amyotrophic lateral sclerosis; (4) unilateral paralysis agitans; (5) a focal lesion either cerebral or spinal; (6) a type of cerebrospinal syphilis; (7) as a peripheral or hysterical affection.

Diagnosis.—The diagnosis of the cause depends upon finding other symptoms of the conditions above mentioned.

3. Progressive Lenticular Degeneration

Progressive lenticular degeneration is a rare disease first described by Kinnier-Wilson. The cells and fibers of the lenticular nuclei gradually disappear, their place being taken by glial tissue. This nucleus probably has something to do with regulating muscle tone (p. 621).

Occurrence and Duration.—It may attack more than one member of a family. It begins in early life and may be acute or chronic, death occurring in from six months to five years.

Symptoms.—The symptoms are bilateral tremor of the intention type affecting all the limbs, spasticity of the limbs and face, dysphagia and dysarthria, sometimes either spasmodic laughing or crying without cause. The reflexes are normal and there is no motor paralysis. There may be some mental weakness, and cirrhosis of the liver is often associated. Very similar symptoms have been described under the name of *pseudosclerosis* (p. 745). Somewhat similar lesions and symptoms may follow poisoning by illuminating gas.

4. Cerebral Palsies of Children

Spastic Paralysis of Children

The paralysis may involve either all four limbs (diplegia), the lower limbs (paraplegia), or the arm and leg of one side (hemiplegia). The face and tongue may also be affected.

They may be either congenital or develop during the first few years of life.

Congenital cases are due either to a failure of the cells in the motor region of the cortex to develop, this type usually occurring in children born prematurely, or to venous meningeal hemorrhage due usually to the use of the forceps, but which may occur in easy labors. These cases have been termed *Little's disease.* They are usually either diplegic or paraplegic.

Those occurring later are due either to hemorrhage from or thrombosis in one of the branches of the middle cerebral artery or inflammation of the cortical cells (encephalitis).[1] They practically always follow either one of the infectious fevers, especially either diphtheria or pertussis and infantile jaundice, and are of the hemiplegic type.

The occurrence of *meningeal hemorrhage* at birth may sometimes be recognized by the occurrence of tense and bulging fontanelles, asphyxia, unequal pupils, slowing of the heart's action, blood cells in the cerebrospinal fluid and muscular rigidity.

Symptoms.—Usually the symptoms are not recognized until the child is old enough to sit up, when it will be noticed that he cannot sit or that he cannot hold up his head, and when the child should walk he does not.

Examination will show more or less muscular rigidity which increases as the child develops, increased reflexes and the Babinski reflex. The spasm of the adductor muscles may be so great as to cause crossing of the legs when walking is attempted (Fig. 173).

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[1] These cases which are due to hemorrhage or inflammation are not true system diseases, as other parts of the brain may be affected. They are described here for convenience.

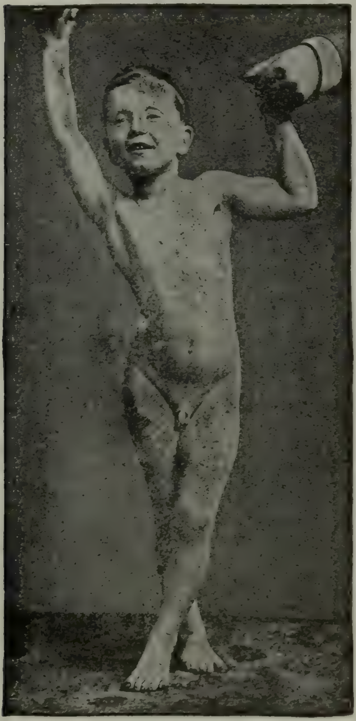

Fig. 173.—Spastic Paraplegia; Cross legged Progression. (Jefferson Hospital; from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

It is in such cases that athetosis (p. 560) is usually found and epileptic convulsions and imbecility frequently but not always coexist.[1]

The above is the usual type but cases have been described in which at first there was noticed muscular hypotonia with consequent abnormal mobility of the joints. As the child grows, ataxia of the cerebellar type and incoördination of the arms is noted. The knee jerks may be increased, diminished, or lost, and the Babinski reflex present or not.

This type has been termed the *atonic ataxic type* of infantile paralysis. It differs from *amyotonia congenita* (p. 534) in the occurrence of the ataxic symptoms and mental deterioration; from *amaurotic family idiocy* in the absence of blindness and the peculiar appearance of the optic nerve found in that disease.

When the paralysis follows an infectious disease, the onset is apoplectic, usually with convulsions, and is apt to occur during the period of convalescence.

The symptoms are those of a spastic hemiplegia, sometimes with involvement of the face and the frequent coexistence of epilepsy, which sometimes is of the Jacksonian type (p. 614), and imbecility. There is usually failure of the affected side to develop equally with the other and the muscles may appear atrophied.

It will be found, however, that the entire limb is smaller, and that the muscles, while small, are firm and that they respond well to the faradic current (Fig. 112).

Conditions to Be Differentiated from the Cerebral Palsies of Children

The cerebral palsies of children must be distinguished from:

Hereditary spastic paraplegia
Acute anterior poliomyelitis
Obstetric paralysis
Multiple neuritis
Rachitis
Chorea.

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[1] As a rule they do not occur in the cases due to arrested development of the motor centers (agenesis).

HEREDITARY SPASTIC PARAPLEGIA.

The symptoms of hereditary spastic paraplegia appear when the child is several years old; they develop gradually. Previous members of the family are apt to have suffered. Epilepsy and mental impairment are not symptoms (p. 686).

ACUTE ANTERIOR POLIOMYELITIS.

Acute poliomyelitis can at once be recognized by the history and the existence of paralysis of the lower neuron type.

OBSTETRIC PARALYSIS.

Obstetric paralysis (p. 661) can be distinguished by the distribution of the paralysis and the fact that it is of the lower neuron type.

MULTIPLE NEURITIS.

Multiple neuritis, following diphtheria especially, may be mistaken for this condition. However, there is no failure of mental development; the paralysis is of the lower neuron type; cranial nerves are frequently attacked and recovery is apt to occur.

RACHITIS.

The muscular rigidity which may occur in rachitis may be mistaken. In this the spasm is confined to the hands and arms; it is intermittent and often painful; laryngismus stridulus may occur.

CHOREA.

Athetoid movements may be mistaken for chorea, but attention to the history of the condition, the presence of increased reflexes, Babinski reflex, the character and location of the movements, will point to the former.

(*b*) *Diseases of the Lower Motor Neurons*

1. Acute Anterior Poliomyelitis

Acute anterior poliomyelitis, while causing symptoms principally referable to the peripheral motor neurons (anterior horns of cord), is not a true system disease. It has been described on page 188.

2. Ophthalmoplegia

Ophthalmoplegia is described on page 638.

3. Bulbar Palsy

Causes.—Bulbar palsy is caused by disease of the nuclei of the motor cranial nerves, from the seventh to the twelfth, inclusive. It may be acute or chronic.

(I) Acute Bulbar Palsy

Causes.—Acute bulbar palsy may be due to hemorrhage from or thrombosis in the vessels supplying the pons and medulla. The posterior inferior cerebellar artery is most frequently affected (p. 712).

Similar causes to those of poliencephalitis superior (p. 636) may be operative here. The condition is then known as *poliencephalitis inferior* (p. 715). They may occur together.

Symptoms.—The symptoms are difficulty in deglutition, articulation and paralysis of the tongue, soft palate and facial muscles developing more or less acutely. Respiratory and cardiac failure may occur.

When due to apoplexy the symptoms are usually unilateral; if inflammatory they are bilateral. The onset is not so sudden and fever, headache, vomiting, and vertigo coexist.

(II) Chronic Progressive Bulbar Palsy

Labioglossolaryngeal Paralysis

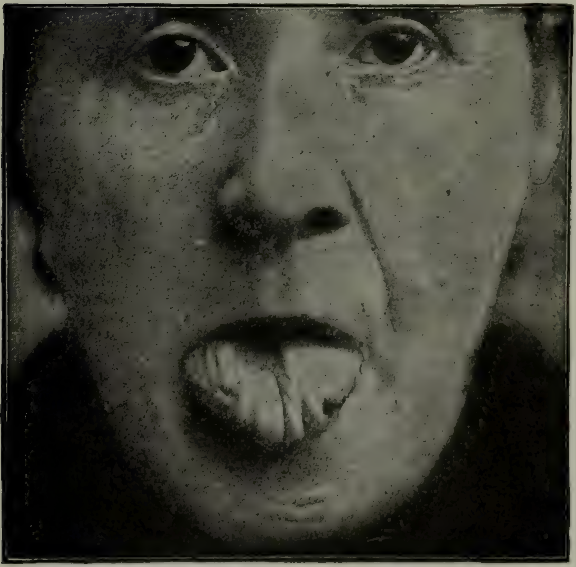

Fig. 174.—Atrophy of the Tongue and Lips in Glossopharyngeal Paralysis. (After Oppenheim; from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

Cause.—This condition is due to degeneration of the nuclei above mentioned.

Occurrence.—It occurs after middle life. Either involvement of the nuclei of the eye muscles or the cells in the anterior horns may occur later or they may occur first (p. 694).

Symptoms.—The first symptom to attract attention is difficulty in articulation, the speech becoming thick, slurred and nasal. First, difficulty in the pronunciation of linguals, then of labials occurs. Swallowing then becomes impaired with regurgitation of food. Semisolids are swallowed best. The tongue cannot be protruded, is tremulous, and atrophied (Fig. 174). The muscles about the angle of the mouth are weak and the muscles about the lines of the face become smoothed out. Fibrillary tremors are present. Saliva drips from the mouth (drooling) and the patient is emotional without due cause.

Conditions to Be Distinguished from Bulbar Palsy

It must be distinguished from:

Pseudobulbar palsy
Myasthenia gravis
Paresis
Thrombosis in the posterior inferior cerebellar artery
Tumor of the medulla
Basal meningitis
Syringobulbia.

PSEUDOBULBAR PALSY.

In pseudobulbar palsy there will be a history of the symptoms following an apoplectic attack (usually two) (p. 708); evidences of paralysis of the central neuron type of the arms and legs will be present and the tongue will not be atrophied.

MYASTHENIA GRAVIS.

Myasthenia gravis (p. 532) is a disease in which the symptoms are due to early exhaustion, and they appear after use of the muscles, to disappear after rest. Peculiar electrical reactions are present (p. 604) and muscular atrophy is not present. Other muscles are affected.

PARESIS.

The disturbances in speech and tremor of tongue and lips may resemble those of paresis (p. 750). In this, however, there is generally Argyll-Robertson pupil, disturbances of the gait, increased or absent knee jerks, the peculiar mental symptoms, and usually evidences of syphilis (increased cell count, Wassermann and globulin reactions) in the cerebrospinal fluid.

THROMBOSIS IN THE POSTERIOR INFERIOR CEREBELLAR ARTERY.

Thrombosis in the inferior cerebellar artery causes bulbar symptoms which are unilateral. They develop suddenly and are accompanied by diminished pain and temperature sense on the opposite side from the lesion and sensory paralysis in the distribution of the fifth nerve, and ataxia on the same side. Paralysis of the cervical sympathetic is often present on the side of the lesion (p. 594).

TUMOR OF THE MEDULLA.

Tumor of the medulla causes bulbar symptoms which, however, are apt to be unsymmetrical in distribution and accompanied by sensory and motor paralysis of the limbs of one or both sides, due to pressure on the sensory and motor tracts. General symptoms of brain tumor (p. 718) will also be present.

BASAL MENINGITIS.

Basal meningitis, syphilitic or otherwise, involves the cranial nerves, but nerves not involved in bulbar palsy are affected, sensory nerves suffering as well as motor, as, for instance, the sixth, seventh, and eighth. The general symptoms of meningitis will also be present.

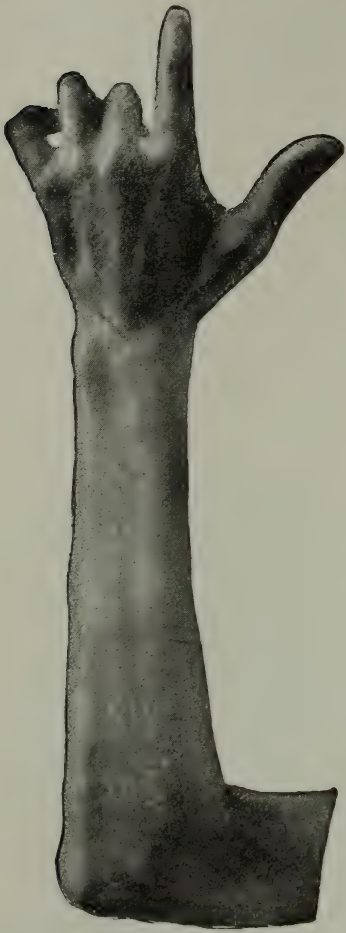

Fig. 175.—Atrophy of the Small Muscles of the Hand and the Muscles on the Extensor Side of the Forearm in Spinal Progressive Muscular Atrophy. The Last Three Fingers Can No Longer Be Extended. (After Strümpell.)

SYRINGOBULBIA.

Syringomyelia, in which the cavity extends into the medulla (syringobulbia), causes bulbar symptoms. They are usually unilateral, and other symptoms of that disease will usually be present.

4. Progressive Spinal Muscular Atrophy

Progressive spinal muscular atrophy (Aran-Duchenne type) is a disease in which there is gradual degeneration and disappearance of the cells in the anterior horns and sometimes of the motor cranial nerve nuclei.

Occurrence.—It usually begins in middle life or after, but may occur in young children and affect a number of persons in the same family (Werding and Hoffman type). Dana has also described a hereditary form appearing in middle life or after.

Etiology.—Some cases of the former type seem to be due to lead poisoning, others to syphilis. It sometimes occurs in those who have had acute anterior poliomyelitis a number of years previously. Many are probably abiotrophies (p. 686).

Symptoms.—The symptoms develop insidiously and atrophy may be present for some time before weakness is noticed. It usually begins in the small muscles of the hand (those supplied by the median and ulnar nerves (pp. 658-659). The peculiar deformity termed *claw-hand* is caused (Figs. 164 and 175).

In rare cases other muscles, as the extensors of the hand, may be first affected. Other muscles, especially those of the forearm, shoulder, girdle and back, sooner or later atrophy and become weak.

Fibrillary tremors are present in the affected muscles and in many of those apparently sound. The tendon reflexes are diminished or absent. The electrical changes are first quantitative decrease, and finally reactions of degeneration (p. 610). The bulbar nuclei may eventually become affected. Sensory symptoms are absent.

Conditions to Be Differentiated from Progressive Spinal Muscular Atrophy

The disease must be differentiated from:

Chronic anterior poliomyelitis
Amyotrophic lateral sclerosis
Syringomyelia
Hemorrhage in the cervical region of the cord
Dystrophies
Progressive neuritic atrophy
Spinal pachymeningitis
Cervical caries
Cervical rib
Arthritic atrophy.

CHRONIC ANTERIOR POLIOMYELITIS.

In chronic anterior poliomyelitis the paralysis is noticed before atrophy; in progressive muscular atrophy the opposite is the case. The paralysis may also develop rather suddenly and then gradually spread to other muscles.

AMYOTROPHIC LATERAL SCLEROSIS.

In amyotrophic lateral sclerosis the knee and tendo Achilles jerks are increased and the legs are more or less spastic.

SYRINGOMYELIA.

Syringomyelia has characteristic sensory symptoms—notably dissociation of sensation (p. 577).

HEMORRHAGE IN THE CERVICAL REGION OF THE CORD.

Hemorrhage into the cord usually follows trauma. The symptoms develop suddenly; paralysis is at first more or less general, followed by improvement.

DYSTROPHIES.

The atrophy in the dystrophies begins in childhood. The shoulder girdle muscles usually suffer first; there may be apparent hypertrophy of some muscles (calves and deltoids), and fibrillary tremors are absent.

PROGRESSIVE NEURITIC ATROPHY.

Progressive neuritic atrophy develops in early life, usually affecting the feet first. Slight pain and diminished sensation may be present.

SPINAL PACHYMENINGITIS.

In spinal hypertrophic pachymeningitis there are shooting pains in the arms and legs, as the case may be; there are often muscular spasms and signs of pressure on the spinal cord.

CERVICAL CARIES.

In cervical caries there will be tenderness of the vertebra and pain in the back on jarring, stiffness of the neck, and usually evidences of pressure on the cord. X-ray examination will show disease of the vertebra.

CERVICAL RIB.

A cervical rib causes paralysis similar in distribution, but usually associated with pain and interference with the circulation. X-ray examination will show it.

ARTHRITIC ATROPHY.

Arthritic atrophy occurs secondarily to inflammation of the joint and is confined to the muscles about that joint.

5. Acute Ascending Paralysis

Landry's Disease

Cause.—This is a progressive paralysis, due to an infection of some sort, which begins in the legs and gradually extends upwards until the muscles of respiration and deglutition are affected, when death ensues. This may occur in forty-eight hours.

Symptoms.—There are no sensory symptoms, the tendon reflexes are lost, but muscular atrophy and change in the electrical reactions do not occur.

The pathological change in most cases is a multiple interstitial neuritis in which the motor fibers only seem to suffer; in others a diffuse myelitis has been found.

Diagnosis.—The rapidity and peculiar way in which the symptoms progress makes the clinical diagnosis clear (the pathological condition present has always been discovered after death).

3. Diseases Affecting Both Upper and Lower Motor Neurons

Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis is a chronic, progressive disease in which there is degeneration of both neurons constituting the motor tract. One may suffer more than the other, causing a preponderance of symptoms peculiar to that neuron.

Occurrence.—It usually commences after middle life.

Etiological Factors.—Lead poisoning, syphilis and other forms of toxemia are etiological factors in some cases; others are forms of abiotrophy (p. 686).

Symptoms.—The symptoms consist of those of progressive spinal muscular atrophy (p. 694) plus spasticity of the legs, increased tendon jerks and the presence of the Babinski reflex. In some cases the atrophy may be confined to the muscles of the hand; in others it may have an extensive distribution. Bulbar symptoms may eventually develop. Sensory symptoms and paralysis of the sphincters are absent.

Conditions to Be Differentiated from Amyotrophic Lateral Sclerosis

The disease must be differentiated from:

Primary lateral sclerosis
Progressive spinal muscular atrophy
Syringomyelia
Cervical pachymeningitis
Transverse myelitis
Combined sclerosis due to anemia.

PRIMARY LATERAL SCLEROSIS.

In primary lateral sclerosis there is no muscular atrophy. Bulbar symptoms never occur.

PROGRESSIVE SPINAL MUSCULAR ATROPHY.

In progressive spinal muscular atrophy the tendon jerks are either diminished or absent. There is no Babinski reflex.

SYRINGOMYELIA.

In syringomyelia there are sensory symptoms consisting of dissociation of sensation (p. 577) in some areas—possibly complete loss in others.

CERVICAL PACHYMENINGITIS.

In cervical hypertrophic pachymeningitis there is severe pain of root type in the arms. The neck is apt to be rigid.

TRANSVERSE MYELITIS.

In transverse myelitis there is apt to be symmetrically diminished or absent sensation below the seat of the lesion. The sphincters are involved and there is difficulty in holding the urine, and unless the lesion is either in the cervical or lumbar region there is no atrophy. There will, in most cases, be a history of acute onset.

COMBINED SCLEROSIS DUE TO ANEMIA.

In the combined sclerosis which occurs in pernicious anemia and other toxic states, the first symptom is intense paresthesia of the extremities. Atrophy does not occur until late in the disease, if at all. The cause, if anemia, will be found by proper blood examination (p. 702).

4. Combined System Diseases

By this is meant a disease which affects both sensory and motor tracts. Tabes dorsalis may be so classified in some instances.

Ataxic Paraplegia, Posterolateral Sclerosis, Progressive Spastic Ataxia

These terms have been applied to conditions in which there are symptoms of spastic paraplegia and ataxia.

Symptoms.—The symptoms are due to degeneration of both the pyramidal tracts and posterior columns. This may be due to several different causes.

Gowers originally applied the name, *ataxic paraplegia,* to what he believed was a primary degeneration of these tracts. Such cases do exist, and Purves Stewart has described a family, several members of which, in three generations, suffered from the symptoms of this condition with the addition of atrophy of the optic nerve, and which he termed hereditary amaurotic ataxic paraplegia. Such cases are *abiotrophies* (p. 686).

Symptoms of ataxic paraplegia may also be caused by a previous transverse myelitis which has improved, but which has caused ascending and descending degeneration of the posterior columns and pyramidal tracts respectively. Syphilis and other toxic conditions cause such symptome of spastic paraplegia and ataxia.

The symptoms, when due to either syphilis, toxemia, hereditary ataxia, or secondary degeneration differ somewhat from those due to primary degeneration, as described by Gowers. They are described on pages 699, 702. The symptoms of the latter develop gradually, and consist of a gait which is a combination of spasticity and ataxia (pp. 586, 686). Difficulty may be experienced in walking in the dark, and the Romberg symptom is present. Muscle hypotonia is not present, as in tabes, the knee and Achilles and possibly the biceps and triceps jerks are increased, and the Babinski reflex is present. Muscle sense may be impaired and incontinence or difficulty in urination may or may not be complained of.

Diagnosis.—Careful inquiry must be made into the existence of the causes mentioned before deciding that it is due to primary degeneration. If due to a previous transverse myelitis, symptoms of that disease will have preceded the development of the secondary condition.

Conditions to Be Differentiated from Primary Forms of Ataxic Paraplegia

Primary forms must be distinguished from:
Tabes dorsalis
Cerebellar tumor
Multiple sclerosis.

TABES DORSALIS.

In tabes dorsalis there will be relaxation of the muscles, absent tendon reflexes, pupillary symptoms and pain.

CEREBELLAR TUMOR.

In cerebellar tumor there will probably be headache, papilledema, and other general symptoms of brain tumor (p. 718). The gait will be more like that due to alcoholic intoxication, and the various symptoms of incoördination due to disease of that part of the brain are present (p. 582).

MULTIPLE SCLEROSIS.

Atypical forms of multiple sclerosis may present similar symptoms. The diagnosis of this disease can only be made by finding nystagmus, pallor of the temporal halves of the optic disks, or other symptoms occurring in multiple sclerosis.

(*a*) *Hereditary Ataxia*

Hereditary ataxia occurs in three forms, viz.: (*a*) *Friedreich's ataxia,* (*b*) *cerebellar ataxia of Marie and Nonne,* and (*c*) the *ataxic paraplegic type.*

1. Friedreich's Ataxia

Friedreich's ataxia, or Friedreich's disease, may exist in a number of members and generations of a family. Sporadic cases, however, occur. A history of alcoholism, epilepsy and other neuroses and syphilis may be found in the ancestors.

Symptoms.—The symptoms usually appear between the ages of six and fifteen years, and are due to degeneration, principally of the pyramidal tracts and posterior columns of the cord. Later other tracts may be affected. The lesion is a gliosis and not a connective tissue sclerosis. It belongs to the class of abiotrophies (p. 686).

The first symptom noticed is a gradually developing incoördination, first of the legs, later the arms. The gait is swaying and resembles that of cerebellar disease more than that of tabes.

The Romberg symptom is usually present, and even with the eyes open, there are swaying, oscillating movements of the head, trunk and limbs, if extended (static ataxia).

When the arms are affected, fine movements, such as buttoning the clothing or picking up small objects, are performed with difficulty. The speech becomes slurring and hesitating, and nystagmus, either spontaneous or elicited by movements of the eyeballs, develops. The optic nerves are normal. The knee jerks are lost but the Babinski reflex is present, excepting possibly in the early stages. In many cases there is constant hyperextension of the great toe and various forms of club feet and scoliosis may develop (Fig. 176).

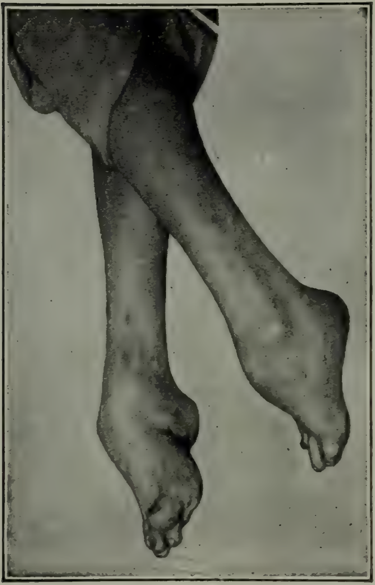

Fig. 176.—Friedreich's Ataxia, Showing the Typical Deformity of the Feet. (After Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

The facial expression is dull. Diminution or loss of sensation, marked motor weakness and muscular atrophy may occur late in the course of the disease.

Conditions to Be Differentiated from Friedreich's Ataxia

This disease must be differentiated from:

Cerebellar type of hereditary ataxia
Hereditary amaurotic ataxic paraplegia
Tabes dorsalis
Juvenile paresis
Multiple sclerosis
Chorea
Cerebellar tumor.

Cerebellar Type of Hereditary Ataxia.

The peculiar features of the cerebellar type are given on page 702.

Hereditary Amaurotic Ataxic Paraplegia.

In hereditary amaurotic ataxic paraplegia there is atrophy of the optic nerve, increased knee jerks, possibly ankle clonus.

Tabes dorsalis.

Tabes dorsalis usually develops later in life, but juvenile cases occur. Argyll-Robertson pupil, characteristic shooting pains, optic atrophy, and positive reaction to the luetin test are symptoms of tabes, while nystagmus, Babinski reflex, the peculiar deformity of the foot and indistinct speech are symptoms of Friedreich's ataxia and not of tabes.

Juvenile Paresis.

Juvenile paresis presents some points in common with Friedreich's ataxia. In the former, however, there will be positive reaction to the various tests for syphilis. The tremor of the hands, tongue and facial muscles is characteristic. The mental disturbances are much more pronounced and nystagmus and deformity of the feet are not present.

Multiple Sclerosis.

Multiple sclerosis, if it appears early, may be difficult to recognize, as there are many similar symptoms; as a rule, it develops later in life. Pallor of the temporal halves of the optic disks, which is a frequent symptom in multiple sclerosis, does not occur in Friedreich's disease; the deformity of the feet, often found in the latter, does not occur in the former. Usually in multiple sclerosis the knee jerks are greatly increased and ataxia in the legs is not present, but marked spasticity is.

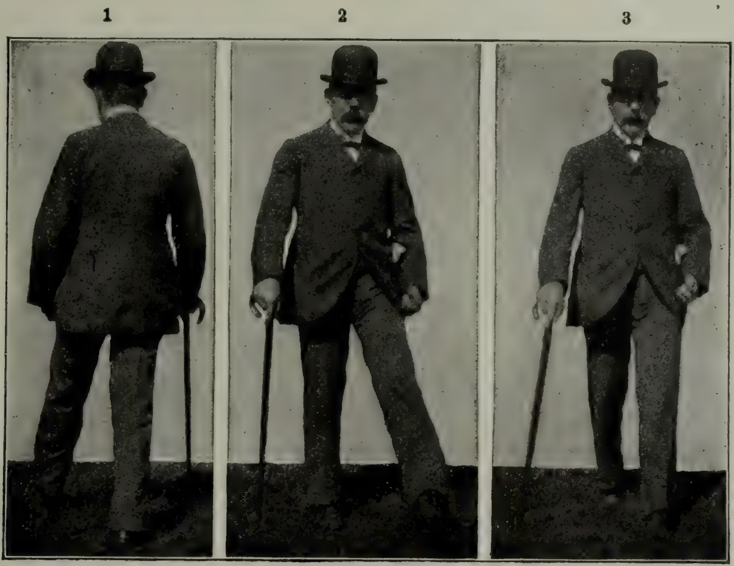

Fig. 177.—The Hemiplegic Gait. 1 and 2, Advancing the Hemiplegic Leg in a Circle from the Hip; 3, Bearing Weight on Paretic Leg and Cane while Advancing Sound Limb. (After Church and Peterson's "Nervous and Mental Diseases," published by W. B. Saunders Co., Philadelphia.)

Chorea.

The peculiar ataxic movements of Friedreich's ataxia may be mistaken for chorea, especially the hereditary form. Choreiform movements occur spontaneously, and not only during movement; nystagmus, Babinski reflex and deformities of the feet are absent and the knee jerk is present.

Cerebellar Tumor.

Cerebellar tumor is distinguished by the existence of headache, papilledema, and other general symptoms of brain tumor, and paralysis of cranial nerves. The symptoms of incoördination (p. 582) are often unilateral.

2. Hereditary Cerebellar Ataxia of Marie

Hereditary cerebellar ataxia of Marie develops usually between sixteen and thirty-five years of age and is due either to an atrophy or degeneration of either the cerebellum or its peduncles and tracts connected with them. It is a rare condition. Sanger Brown, of Chicago, has described a series of cases.

The symptoms resemble very much those of Friedreich's ataxia, except that there are no deformities, the knee jerks are increased and there is optic atrophy. Mental deterioration may or may not be present.

Differentiation.—The diseases with which it is likely to be confounded and the differential points are given in the table on page 703.

3. Hereditary Amaurotic Ataxic Paraplegia

This condition is referred to on pages 698, 703.

(*b*) *Combined Sclerosis of the Spinal Cord*

Combined sclerosis of the spinal cord, also termed *subacute combined sclerosis* by Russell, Batten and Collier, and *diffuse degeneration of the spinal cord* by Putnam and Taylor, is most frequently found in cases of pernicious anemia. It is, however, often due to other toxemic and exhausting conditions, as influenza, chronic diarrhea, lead poisoning, pellagra and malaria. Anemia may be secondary to these causes.

The posterior columns and lateral tracts are degenerated, and in some cases other tracts and the cells in the anterior horns may be also affected.

Symptoms.—The symptom first noted is intense paresthesia in the feet followed by some ataxia, and possibly muscular weakness; after a time the hands and arms become similarly affected. At first the limbs are more or less spastic, the tendon jerks are increased and Babinski's reflex is present. Motor weakness gradually becomes marked and sensory loss may be present, as may pain in the limbs and girdle sensation; the movements of the arms and hands are ataxic. As the disease advances spasticity disappears and the limbs become flaccid, the knee jerks disappear and muscular atrophy with reactions of degeneration may occur. Loss of control of the sphincters also appears during this stage, as may either delirium or the development of dementia. Fever may be present. Symptoms following this course and associated with any of the causes mentioned make the diagnosis comparatively easy.

Symptoms	Friedreich's Ataxia	Hereditary Amaurotic Ataxic Paraplegia	Tabes dorsalis	Paresis	Multiple Sclerosis	Hereditary Chorea	Cerebellar Tumor	Hereditary Cerebellar Ataxia
Reflexes	Tendon jerks absent; Babinski present	Tendon jerks usually increased; Babinski present usually	Tendon jerks and Babinski absent	Tendon jerks either increased or absent	Tendon jerks increased. Babinski present	Normal	Tendon jerks increased or absent. Babinski may or may not be present	Knee jerks increased or normal. Ankle clonus sometimes present; Babinski absent
Eyes	Nystagmus	Optic atrophy. Divergent strabismus due to blindness	Possibly optic atrophy, double vision, usually Argyll-Robertson pupils	Same as tabes	Nystagmus; pallor temporal halves of the disks; sometimes paralysis ocular muscles	Normal	Nystagmus; papilledema; paralysis ocular muscles sometimes	Nystagmus; atrophy of optic nerve; sometimes diplopia
Speech	Slurring and hesitating	No change	No change	Slurring, hesitating	Scanning usually; sometimes slurring	Slurring, indistinct	No change	As in Friedreich's disease
Sensory	Areas of sensory loss rarely	Normal	Pain, areas of diminished or lost sensation	No change usually	Rarely areas of lost or diminished sensation	Normal	Normal unless pons involved	Usually none
Deformities	Hyperextension of great toe. Pes cavus scoliosis	Talipes equinovarus in some cases	Enlargements of joints sometimes	Usually none	None	None	None	None
Incoördination	Arms and legs. Romberg symptom	Ataxia upper limbs usually. Legs ataxic and spastic	Ataxic gait usually. Upper limbs not affected. Romberg present	Legs may be either ataxic, spastic or combination of the two	Intention tremor; usually no other evidence of incoördination. Legs spastic	None. Gait may be hesitating and unsteady	Cerebellar gait; dysmetria adiadochokinesia (p. 582)	Of cerebellar type
Age at onset	6 to 15 years usually	Infancy to 7 yrs.	Usually 30 to 40; may rarely develop in early life	Same as tabes	About 30; may be younger or older	30 to 40 years	Any age	16 to 30 years
Occurrence in more than one member of a family	Hereditary sporadic cases occur	All 3 cases reported occurred in one family	Does not unless syphilis is present, when it might	Same as tabes	Does not as a rule	Hereditary	Does not, except by coincidence	Hereditary; sporadic cases may occur
Mental	May be some deterioration later; often none	None	None except late in disease	Marked dementia, usually delusions of grandeur	Often emotional; may be mental deterioration late	Mental deterioration	None	May be none. Sometimes mental deterioration

Conditions to Be Differentiated from Combined Sclerosis of the Spinal Cord

The following conditions may be mistaken for this disease:

Multiple sclerosis
Ataxic paraplegia due to other causes
Multiple neuritis
Acute myelitis
Tabes dorsalis
Tumor of the cord.

Multiple Sclerosis.

The atypical form of multiple sclerosis may at first be difficult to distinguish. The paresthesia is not so great, if present at all, and some other symptoms, as atrophy of the temporal halves of the optic disks, may be present. Typical cases of multiple sclerosis should present no difficulty. The previous existence of primary anemia or any of the causes mentioned will be in favor of combined sclerosis.

Ataxic Paraplegia.

In ataxic paraplegia due to other causes (p. 698) the intense paresthesia is absent.

Multiple Neuritis.

The flaccid stage may simulate multiple neuritis. The previous condition of spastic paralysis and the absence of tenderness over the nerve trunks are not characteristic of neuritis.

Acute Myelitis.

The paralytic symptoms of acute myelitis develop more suddenly; pain and paresthesia are not so great, if present at all. Sensory paralysis is more sharply defined, and the action of the sphincters is disordered early.

Tabes dorsalis.

Tabes dorsalis may be simulated somewhat by the flaccid stage. The previous history of spastic paralysis, the greater motor weakness, and the absence of Argyll-Robertson pupil and presence of Babinski's reflex distinguish combined sclerosis.

Tumor of the Cord.

The absence of root pains and the early diffuse distribution of motor and sensory symptoms exclude spinal tumor.

G. Diffuse and Focal Diseases of the Brain

1. Affections of the Blood Vessels

Blood Supply of the Brain

The external carotids furnish the blood to the scalp, skull, and dura mater. The middle meningeal is the most important branch, as it is frequently ruptured by traumatism (*infra*).

The internal carotids and vertebrals furnish blood to the pia mater and brain. The former divides into the anterior and middle cerebrals, which are united by the anterior communicating artery. The vertebrals unite to form the basilar which divides into the posterior cerebrals; these are united with the middle cerebrals by means of the posterior communicating arteries, thus forming the *circle of Willis.* An important branch of the vertebral artery is the posterior inferior cerebellar. From the circle of Willis are given off branches known as *central arteries* which supply the basal ganglia and adjacent white matter. They are *end arteries.* Branches from the middle cerebral supply the posterior two-thirds of the posterior limb of the internal capsule and corpus striatum. They are frequently the source of cerebral hemorrhage (apoplexy). The anterior third of the posterior limb of the internal capsule is supplied by the posterior communicating. The optic thalamus, corpora quadrigemina and crus cerebri are supplied by branches of the posterior cerebral.

The three cerebral arteries are distributed over the cortex in the pia mater and supply it and the underlying white matter. They are known as *cortical arteries* and anastomose more or less with each other.

The *anterior cerebral* supplies most of the frontal lobes and upper part of the sensorimotor region; the *middle cerebral* the precentral convolution except the upper part (*supra*), the third frontal, island of Reil, and part of the temporal and parietal lobes.

The *posterior cerebral* supplies the occipital lobe and part of the temporal. The pons and cerebellum are supplied principally by branches from the *basilar;* the medulla and part of the cerebellum by branches from the vertebrals, principally the posterior inferior cerebellar arteries. The veins empty into the various sinuses.

(*a*) Meningeal Hemorrhage

Meningeal hemorrhage is rarely spontaneous except when it occurs as *hemorrhagic internal pachymeningitis.*

Hemorrhage due to traumatism, usually a blow on the head or face, is comparatively common. The *middle meningeal* is the vessel most commonly ruptured. The clot then is extradural.

Symptoms.—The usual history is that after the blow, which may or may not cause some symptoms of concussion, there is an interval varying from a few minutes to a number of hours in which there are no symptoms; then the patient becomes gradually comatose. There may or may not be muscular twitchings, rigidity and some paralysis of the limbs on the opposite side. The pupil upon the affected side is frequently dilated (Hutchinson's pupil). Other irritative or paralytic symptoms may be present, depending upon the location of the hemorrhage. Some swelling of the optic nerve (papilledema) may be noticed on the affected side. The pulse is usually slow and the blood pressure high.

When the hemorrhage is *subdural* the symptoms usually develop quicker and the paralysis is greater and more permanent. Blood will also be found in the cerebrospinal fluid removed by lumbar puncture.

Intracerebral hemorrhage may also be associated, in which case the symptoms will depend on its location (*infra*).

(*b*) *Apoplexy* .

Apoplexy is a term used to denote sudden paralysis, often accompanied by unconsciousness, and due to a lesion of the vascular system of the brain.

The different **lesions which may cause this condition are:**

1. Hemorrhage due to the sudden rupture of a vessel.
2. Blocking up of a blood vessel by:
   (*a*) Embolus.
   (*b*) Formation of a thrombus.
3. Temporary closure of an artery due to either spasm or lacunar degeneration.

The second cause results in acute cerebral softening.

Apoplexy is a disease usually occurring either in *early life* or during the *degenerative* period. Thomas gives the following table, showing the **relative liability** at different periods of life:

Decade	*Cases*
First	135
Second	25
Third	62
Fourth	102
Fifth	137
Sixth	143
Seventh	105
Eighth	25
Ninth	6
	740

The causes of the condition during the first decade have been detailed on page 689, and will not here be considered. Meningeal hemorrhage and traumatic apoplexy are described on page 705. The following, therefore, deals with cases in which the lesion is intracranial and not due to traumatism, and in which the time of onset is after the first decade.

Apoplexy when not due to embolism, is always preceded by arterial disease (endarteritis, periarteritis, atheroma, fatty degeneration).

The usual causes of arteriosclerosis are either alcoholism, syphilis, or lead poisoning. Some people, however, are born with a tendency to early degeneration of the arteries and such cases may be classed among the abiotrophies (p. 686).

Fatty degeneration is usually due to one of the infectious fevers but may be due to such conditions as purpura, scurvy, leukemia and marasmic conditions.

1. Hemorrhagic Apoplexy

Occurrence and Etiology.—Hemorrhagic apoplexy usually occurs after the fortieth year, and is liable to occur at a much earlier age than acute softening due to thrombosis. Cases occurring before forty are usually due either to syphilis, embolism, or fevers.

Physical exertion that increases blood pressure predisposes to an attack. The parts affected in the order of frequency are: Corpus striatum and internal capsule; cortex and subcortex; optic thalamus; cerebellum; pons and medulla. Ventricular hemorrhage is usually secondary to one within basal ganglia or capsule.

Symptoms.—The attack is always sudden (except in ingravescent apoplexy, p. 708) and consciousness is usually lost at once. If it is in or near the motor cortex convulsions may occur, but this is not the rule in adults while it is in children. The face is flushed, pulse slow, blood pressure high and breathing usually stertorous. The pupils are contracted, sometimes unequal and irresponsive to light. The temperature just after the attack may be below normal, followed later by a rise above. This is more marked on the paralyzed side if hemiplegia is present. The urine may be retained or evacuated involuntarily. Some swelling of the optic disk may also be present on the affected side. If, as is usually the case, the corpus striatum and internal capsule are the seat hemiplegia on the side opposite the lesion at once develops. This can be detected by noting that the mouth is drawn to the opposite side, the Babinski reflex is present, and that the limbs when lifted seem more relaxed than those of the other side. In some cases the affected limbs may be rigid and the tendon jerks lost (early rigidity due to irritation).

Conjugate deviation of the eyes (p. 636) at first, in some cases, away from the side of the lesion due to irritative spasm of the muscles, but later, and sometimes from the first, to the side of the lesion, is usually present.

Hemianesthesia may be present and if the posterior part of the capsule is involved may remain; if not, it soon disappears.

If death does not occur, consciousness returns in twenty-four to forty-eight hours, with difficulty in speech due either to aphasia (p. 614) or to paralysis of the tongue and muscles concerned in articulation; if due to the latter it soon improves. There will also be difficulty in swallowing, which usually soon disappears (*infra*). The tongue when protruded deviates to the paralyzed side (p. 653). The muscles about the angle of the mouth are paralyzed (incomplete facial palsy, p. 642).

If recovery from the attack occurs the usual history is a greater or less degree of permanent paralysis of the hemiplegic type with the development of contractures, increased tendon jerks, and the Babinski reflex. When walking the leg is swung in the arc of a circle and the hip elevated. The usual attitude of the patient is shown in Figure 177.

Epileptic convulsions may occur at intervals, but this is not the rule in adults as it is in children. The various spasmodic conditions common in the cases arising in childhood (p. 560) are rare. Mental impairment may or may not develop. The limbs may be cyanotic and cold. Sometimes severe pain is experienced in the paralyzed side. In such cases a lesion of the optic thalamus has been found (p. 621).

Ordinarily the power of articulation (unless the speech centers are affected) and swallowing returns. In the exercise of these functions the muscles of each side habitually act together and hence can be innervated from either side of the brain. If, however, a patient has two separate attacks, one involving one side and the other the other side of the brain, or if the association fibers between the two sides are cut off by one attack, these functions are more or less permanently interfered with and the condition known as *pseudobulbar palsy* results (See p. 692).

Hemorrhage involving the *lenticular nucleus* alone causes marked hypertonicity of the affected side with slight or no motor paralysis and absence of the Babinski reflex. If this nucleus is not interfered with, the lesion being confined to the pyramidal tracts, there may be flaccidity. If both the extrapyramidal and pyramidal tracts are destroyed the paralysis is flaccid and recovery of power does not occur. (See pp. 556, 558, 621).

Hemorrhage into the ventricles may be primary, but is usually secondary to hemorrhage in their vicinity; death soon occurs in primary cases. In secondary ones it may be suspected if, after apparent improvement, there is a sudden return and increase of the symptoms and death soon occurring.

Ingravescent apoplexy is due to a hemorrhage first into the external capsule, which then breaks through the white matter into the lateral ventricle. At first the patient has merely vertigo, headache and vomiting; then hemiplegia gradually develops, followed by loss of consciousness and death. If the *optic thalamus* is the seat of the hemorrhage the symptoms given on page 621 result.

Hemorrhage into the pons may be known by the sudden development of *marked* contraction of the pupils, slow respiration, high temperature, unconsciousness, and crossed paralysis (p. 622, Fig. 137).

Hemorrhage into other localities may be suspected by the sudden development of unconsciousness and symptoms referable to the region involved and its neighborhood (See Cerebral Localization and Cerebral Softening).

Conditions to Be Differentiated from Hemorrhagic Apoplexy

Cerebral hemorrhage must be distinguished from:

Coma, due to concussion or compression of the brain
Alcoholic coma
Uremic coma
Diabetic coma
Opium poisoning
Stuporous condition following epilepsy
Hysteria
Comatose state in pernicious malarial fever
Gas poisoning
Apoplectiform seizures occurring in paresis, brain tumor, and multiple sclerosis
Acute cerebral softening
Arterial spasm.

COMA.

In all cases of coma without history careful search must be made for evidence of head injury as fracture of the skull. The possibility of meningeal hemorrhage (p. 705) must be borne in mind. In concussion evidences of shock will be present; localized paralysis will be absent.

ALCOHOLIC COMA.

Alcoholic coma is characterized by the odor of alcohol on the breath; but it must be remembered that this may be due to one drink as well as many, and, therefore, may be present in a victim of apoplexy. In it, however, the patient may often be aroused; there will be no evidence of paralysis; the Babinski reflex, contracted and irresponsive pupils, and conjugate deviation of the eyes will be absent.

UREMIC COMA.

This may be difficult, especially in cases of uremia in which hemiplegia or other focal paralysis occurs. This, however, is usually more transient than that due to hemorrhage. The odor of urine upon the breath and the presence of pregnancy would point to a toxemic origin of the symptoms. If there is no paralysis, sudden coma is more commonly due to uremia than hemorrhage. Preceding convulsions are more likely to occur in uremia. It must not be forgotten that diseased kidneys are common in those liable to apoplexy, but it is usually of the interstitial type; therefore, symptoms as edema, a large amount of albumen and casts in the urine, and albuminuric retinitis, point to uremia, but there are exceptions to this.

Diabetic Coma.

In diabetic coma there will be a fruity odor of the breath; the urine will be characteristic of diabetes with excess of acetone and diacetic acid; the pulse rapid and of low tension. Paralysis is not present.

Opium Poisoning.

Opium poisoning somewhat resembles pontile hemorrhage; in the latter, however, the temperature is high and localized paralysis present, which are not symptoms of the former.

Stuporous Condition Following Epilepsy.

Stupor following an epileptic attack usually clears up in a short time. Evidences of having bitten the tongue may be found. Localized paralysis is not present.

Hysteria.

Hysterical coma and paralysis will be described on pages 776, 785.

Comatose State in Pernicious Malarial Fever.

Pernicious malaria is rare in temperate regions. High temperature and examination of the blood will indicate it if suspected.

Gas Poisoning.

In this there is cyanosis; the odor of gas may be detected. Localized paralysis is not present.

Apoplectiform Seizures Occurring in Paresis, Brain Tumor, and Multiple Sclerosis.

In paresis, brain tumor, and multiple sclerosis apoplectiform seizures may occur. These are probably due to sudden congestion and are usually transient. The diagnosis will depend upon the history and finding other symptoms of the respective diseases present.

Acute Cerebral Softening—Arterial Spasm.

Acute softening and arterial spasm are considered on pages 711, 713.

2. Acute Cerebral Softening Due to Embolism

Occurrence.—Acute cerebral softening due to embolism occurs most frequently between the ages of twenty and fifty years.

Causes.—The predisposing causes are: endocarditis, acute infectious diseases; anemia; pregnancy; blood dyscrasias and malaria. In malaria the embolus consists of the parasites and their products. Softening and degeneration of the nerve fibers and cells in the area supplied by the plugged vessel result. If an embolus is derived from an infected focus, as ulcerative endocarditis, abscess may form.

Onset.—The onset is sudden either with or without loss of consciousness. If the motor area or tract is affected hemiplegia results and as, the left side of the brain is the more frequently affected, aphasia is often caused. More or less permanent paralysis as in cerebral hemorrhage results.

Differentiation.—In the absence of history, coma, when it is present, must be distinguished from that due to other causes (p. 709). The differential diagnosis from hemorrhage will be found on page 714.

3. Acute Cerebral Softening Due to Thrombosis

Occurrence.—Acute cerebral softening due to thrombosis occurs most commonly between the ages of fifty and seventy, and is probably the most frequent cause of apoplectic attacks. It may occur during early adult life if due to syphilis. Apoplexy due to syphilitic disease of the arteries is usually thrombotic.

Causes.—The predisposing causes are syphilis, lead, gout, weakness of the heart muscle, blood dyscrasias.

Symptoms.—Similar changes in the brain substance occur, as in cerebral embolism.

Before the development of a thrombus prodromes frequently occur. These consist of headache, vertigo, numbness of the hands and feet and drowsiness. The onset may be sudden, but in a large proportion of cases is gradual, it requiring from a few minutes to several hours before paralysis becomes complete. This is due to the gradual development of the thrombus and progressive involvement of different vessels. Consciousness is usually preserved, although the patient may be stuporous; if lost it becomes so gradually. The attack is apt to occur during sleep, while the heart's action is weak. The blood pressure may be increased, but is often low and the rapidity of the heart beats increased. There are no signs of excitement of the circulation, and temperature changes are slight if they occur.

If the vessels supplying the motor area are involved, hemiplegia occurs. The resulting condition is similar to that due to hemorrhage (p. 708). The outlook for improvement is better. Vessels supplying other areas may be affected as in hemorrhage (See p. 712). After branches of the middle cerebral it occurs most frequently in the posterior inferior cerebellar branch of the vertebral, basilar, and posterior cerebral.

Occlusion of the posterior inferior cerebellar artery causes the following *symptoms* which develop suddenly without loss of consciousness: On the side of the lesion paralysis of the soft palate, vocal cords and muscles of deglutition causing difficulty in swallowing and hoarseness of the voice; signs of paralysis of the cervical sympathetic (p. 594), loss of sensation for pain and temperature in the distribution of the fifth nerve, ataxia of cerebellar type and tendency to fall to this side. On the opposite side there may be slight motor weakness, diminution of sensibility for pain and temperature, slight ataxia.

Rarely there may be some of the following symptoms: Deafness and tinnitus, paralysis of the face, tongue, external rectus muscle of the eye, diminished sense of taste on the side of the lesion, and headache, hiccough, nausea, vomiting, and disturbance of micturition.

Occlusion of the superior cerebellar artery probably causes ataxia of the limbs on the same side, loss of pain and temperature sense, power of emotional expression, and deafness on the opposite side.

In *thrombosis in the basilar artery* the pons is affected, and crossed paralysis and other symptoms of lesions there are present (See also Cerebral Localization, p. 611).

Conditions to Be Differentiated from Acute Cerebral Softening Due to Thrombosis

Apoplexy due to thrombosis must be distinguished from that due to hemorrhage, spasm, and lacunar degeneration; also from:

Cerebral syphilis
Encephalitis
Hysteria
Tumor of the brain.

CEREBRAL SYPHILIS.

The development of the symptoms in cerebral meningitis due to syphilis may be acute. There will usually, however, be violent headache; consciousness is not completely lost, and paralysis, if present, is not so marked as would be due to apoplexy (See p. 747). The cerebrospinal fluid will probably contain an increased number of lymphocytes and other evidences of syphilis (p. 758).

ENCEPHALITIS.

Encephalitis may involve the motor region alone and cause hemiplegia of rapid onset. It may also involve other regions, especially the bulbar nuclei (pp. 689, 715). It usually follows an infectious fever and is associated with headache, fever, and symptoms of meningeal irritation. If the bulbar nuclei are also affected the probabilities are in favor of encephalitis.

HYSTERIA.

The peculiarities of hysterical hemiplegia are described on page 785.

TUMOR OF THE BRAIN.

In rare instances the thrombosis may develop very gradually, spreading from vessel to vessel and causing a paralysis which may take weeks or months to develop. Such cases may simulate brain tumor in the motor region (p. 720). If the symptoms occur in a young person the probabilities are in favor of tumor. If the general symptoms (p. 718) of tumor are present the diagnosis should not be difficult, but they may be absent. Tumor is also more likely to so develop than thrombosis.

4. Apoplexy Due to Arterial Spasm—Cerebral Claudication

Degenerating arteries are liable to spasm; when this occurs in cerebral arteries, apoplectiform symptoms result. Consciousness is not lost and the symptoms usually disappear in from a few hours to a few days.

Transient attacks of apoplexy may occur in paresis, multiple sclerosis, and brain tumor, but other symptoms of these diseases will be found (p. 807).

5. Lacunar Degeneration

Lacunar degeneration is characterized by small cavities scattered throughout the brain, through which runs a sclerotic artery. The cavities are believed to be due to lack of nutrition. These may lead to attacks of hemiplegia or other forms of paralysis of a more or less transient nature. This process may also occur in the spinal cord.

The table on page 714 gives the usual characteristics of the attack due to each of the several causes mentioned. It is important to recognize the nature of the cause if possible for therapeutic reasons. In some cases it may be impossible.

6. Thrombosis of the Cerebral Sinuses

Thrombosis of the cerebral sinuses is usually inflammatory and occurs secondarily to suppuration in the middle ear. In such cases there are symptoms resembling those of meningitis. They may be associated.

If the *lateral sinus* is affected there will be swelling and edema over the mastoid and surrounding region, tenderness and hardness over the jugular vein. Involvement of the *longitudinal sinus* causes swelling of the frontal and parietal veins. In thrombosis of the *cavernous sinus* there is exophthalmos, orbital edema, swelling of the frontal veins, paralysis of the motor nerves of the eye, and sometimes of the fifth nerve.

The condition can be distinguished from meningitis by examination of the cerebrospinal fluid, which will be normal. Cerebral abscess (*vide*) may be associated.

	Cerebral Hemorrhage	Cerebral Thrombosis	Cerebral Embolism	Arterial Spasm and Lacunar Degeneration
Onset	Very sudden	Usually gradually, 1 to 3 hours, may be weeks, rarely	Sudden	Sudden
Time of Onset	During muscular effort	During the night	Any time	Any time
Age at Onset	40 to 50 yrs. May occur in childhood	After 50 yrs. May occur in childhood or early adult age if due to syphilis or infectious fevers	Usually in young adults	Old age
Predisposing Cause	Arterial degeneration	Arterial degeneration; especially if associated with weak heart	Some cause for embolism	Arterial degeneration
Consciousness	Lost at once	Often not lost; may be so gradually or only stuporous	May or may not be lost	Not lost
Temperature	Initial fall followed by rise; more marked on side of lesion	Usually no change	No change	No change
Circulation	Pulse slow, full. Blood pressure high	Pulse rapid, often weak. Pressure either high or low	Pulse rapid; no special change in pressure	Pulse may be slow or rapid. Blood pressure varies.
Duration of Attack	Unconscious several hours to a day or more. Paralysis more or less permanent.	Recovery of power more apt to occur than in hemorrhage. Usually some permanent impairment	Same as thrombosis	Improvement rapid. Usually complete disappearance of symptoms
Eyes	Papilledema on affected side. Contracted, often unequal pupils. Conjugate deviation	May be conjugate deviation	May be conjugate deviation	No change

Thrombosis may also be non-inflammatory in origin and is then due to weak heart or general exhaustion. It occurs most frequently in either infants or old people. Constitutional symptoms will be absent.

(*c*) *Inflammation of the Brain*

(*Encephalitis*)

1. Acute Hemorrhagic Encephalitis

Causes.—Acute hemorrhagic encephalitis is due to an infection of some sort. The chief causes are the acute infectious diseases, especially influenza, and chronic alcoholism. Mineral poisons, especially lead, and ptomain poisons may rarely cause it. It is more common in the young.

Symptoms.—The lesions consist of scattered foci of inflammation, which do not suppurate, scattered throughout the brain. The symptoms, therefore, vary according to the distribution of the lesions.

Certain areas are more susceptible, viz., the gray matter of the cortex, especially in the motor region (poliencephalitis of Strümpell), and the gray matter about the aqueduct of Sylvius, with involvement of the nuclei of the motor nerves of the eyeball (poliencephalitis superior of Wernicke, p. 636). The nuclei of other motor cranial nerves (poliencephalitis inferior, p. 692) and the cerebellum may be affected. The lesions may be confined to either one or any combination of these regions.

Onset.—The onset of paralysis is usually preceded by headache, vomiting, slight rigidity of the neck, vertigo. These symptoms may be followed by more or less stupor. Fever may or may not be present, leukocytosis and optic neuritis usually are. If confined to the *motor region of the cortex* there will be hemiplegia and possibly Jacksonian convulsions. This is one of the causes of cerebral palsy in children (p. 689). If the cerebellum is involved cerebellar ataxia will be present (p. 582). The symptoms of poliencephalitis superior and inferior are described on pages 637, 692. The spinal cord may also be affected, causing symptoms of acute poliomyelitis (polioencephalomyelitis).

Conditions to Be Differentiated from Acute Hemorrhagic Encephalitis

Encephalitis must be distinguished from:

Meningitis
Cerebral apoplexy
Brain tumor.

MENINGITIS.

In meningitis the symptoms of irritation of the meninges (hyperesthesia, rigidity) are more marked. The cerebrospinal fluid will be more cloudy and contain an increase in polymorphonuclear cells and the pathogenic germ.[1] In encephalitis the fluid is likely to be clear and contain nothing but an increased number of lymphocytes. Paresis is a form of meningo-encephalitis (See p. 749).

CEREBRAL APOPLEXY.

Cerebral apoplexy is not apt to be mistaken except when it follows an acute infectious fever and is confined to the motor cortex. In such cases the presence of the constitutional symptoms detailed above would be in favor of encephalitis.

BRAIN TUMOR.

The acute onset distinguishes encephalitis from brain tumor. The resulting state, if it consists of hemiplegia and Jacksonian convulsions, may be mistaken at a later period. The history of an acute onset with constitutional symptoms is characteristic of encephalitis.

2. Acute Suppurative Encephalitis

(Abscess of the Brain)

Etiology.—Acute suppurative encephalitis, or abscess of the brain, is due usually to the transmission of infection from either a neighboring part by continuity or a distant part through the blood vessels.

The chief *causes* in the order of their importance are:

Extension from suppurative disease of the middle ear and mastoid cells—in these cases inflammation of the meninges, beginning in the dura, may precede the development of abscess, or the infection may reach the brain through the sinuses; traumatism to the head, usually associated with fracture of the skull; remote septic processes, as ulcerative endocarditis, tuberculosis of the lungs, fetid bronchitis, empyema, abscess of the liver, ozena due to caries of ethnoid and nasal bone, caries of orbital bones, and pyemia; the acute infectious fevers, especially influenza. Most cases occur in males between ten and thirty. Abscesses due to ear disease occur in either the temporal lobe or cerebellum. From any cause they are found most frequently in either the frontal or temporal lobes. They may be single or multiple, usually the latter.

The *symptoms* may be acute or chronic. If the former, trauma is the usual cause, and there would be following this:

Headache, vomiting, chills, fever, mental dullness and delirium plus the focal symptoms attributable to the region in which it is located.

Abscesses, however, are liable to occur in silent regions, and focal symptoms may be absent. Optic neuritis or papilledema may be present and the pulse is usually slow (60-70). Coma develops before death.

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[1] This is not so in tubercular meningitis (See p. 89).

Chronic Form.—The chronic form usually follows suppuration in the middle ear. The abscess may remain latent for a long time. During this period, headache, vertigo, and mental depression may be suffered from; nausea, vomiting and convulsions may also occur. The temperature may be either normal or subnormal. Focal symptoms (p. 611) develop if it is located in a region where such can be caused. Later symptoms such as occur in the acute form may develop, or if it rupture, those of acute meningitis result. Phlebitis and sinus thrombosis (p. 713) may coexist.

Diagnosis.—The diagnosis depends upon the occurrence of these symptoms in association with one of the causes mentioned. It is often difficult.

Conditions to Be Differentiated from Acute Suppurative Encephalitis

It must be distinguished from:

Meningitis
Brain tumor.

Meningitis.

In meningitis the pulse is usually rapid; a slow pulse with elevated temperature is characteristic of abscess. The cerebrospinal fluid presents the characteristics of meningitis; in abscess it is negative. In meningitis due to syphilis an increased number of lymphocytes and the Wassermann reaction may be found in the fluid, and the symptoms will probably improve with proper treatment.

Brain Tumor.

Brain tumor might be confounded with the chronic form and it may be impossible to distinguish the two except by noting the presence of one of the causes of abscess.

(*d*) *Brain Tumor*

Etiology.—These may arise from within the brain or spring from the meninges and cause cerebral symptoms by pressure. Traumatism of the head may certainly be a cause, others may be due to metastasis from a malignant growth elsewhere. In most cases no cause can be determined.

Forms.—All forms of morbid growth may occur—those most common in the order of frequency are: Tubercular, sarcomata, glioma, gliosarcoma, cyst, carcinoma and gumma. If possible, determination of the nature of the growth is of importance.

Tubercular Tumors.—Tubercular tumors may be single or multiple; they are most common in childhood; they may be primary, but are usually secondary to tubercular disease elsewhere. They usually spring from the meninges, but may be within the brain substance. Tubercular meningitis may coexist.

SARCOMATA.—Sarcomata are often primary and usually are encapsulated, rarely infiltrating the brain substance.

GLIOMA.—Glioma infiltrates the brain substance and to the naked eye resembles it closely. The symptoms often develop slowly, and general symptoms are often absent. They are vascular and may be the seat of hemorrhages; they are also apt to break down and form cysts.

GLIOSARCOMA.—Gliosarcoma resembles glioma.

CYSTS.—Cysts are usually due to cystic degeneration in a glioma or gliosarcoma. They may be due to echinococcus infection or to a previous apoplexy.

CARCINOMA.—Carcinoma is due to metastasis, may be single or multiple, and infiltrates the brain substance.

GUMMA.—Gumma arises in the meninges and is usually located at the base (See p. 745).

Symptoms.—The symptoms are both general and focal. *General symptoms* are those which occur independently of the location of the growth. *Focal symptoms* are those due to interference with the functions of the brain centers or the tracts which run to or from them.

General Symptoms.—The general symptoms consist of headache, general epileptiform convulsions, papilledema and consecutive atrophy, changes in visual fields, mental symptoms, projectile vomiting, vertigo, apoplectiform attacks, syncope, insomnia, polyuria, menstrual disturbances.

Some of these are usually present, but any or all may be absent. They depend in severity upon the nature and location of the growth; they are apt to be especially severe in tumors below the tentorium—for instance, in the cerebellum. They also depend in severity upon the rapidity of growth and vascularity of the tumor—thus they may be more or less severe while it is growing; if this cease for a time they may improve very much.

Vascular tumors are liable to hemorrhage within and about them, which causes apoplectiform symptoms. Hyperemia may also cause an increase in other symptoms.

Headache is one of the most constant, but may be absent. It depends either upon the increase in intracranial pressure and stretching of the membranes, or upon involvement of the meninges themselves. It may be constant or intermittent, and is increased by excitement or mental strain; it may be worse at night, especially in gummata. If near the cortex there may be tenderness over the seat of the growth.

Epileptiform convulsions of the general type may be caused by a growth anywhere in the brain. They may be early symptoms, especially in children. They must not be confounded with Jacksonian attacks (pp. 767-768). Special attention must be paid to the nature and location of the aura, as it may have some localizing value.

Papilledema is an important symptom when present; it is so in about eighty per cent. It is most frequent and severe in those near the base. It must be remembered that it may be due to other causes (p. 599). Marcus Gunn states that it may aid in localization to the following extent:

1. An intense double papilledema with much swelling and surrounding retinal change, developing suddenly, suggests the cerebellum.
2. One-sided papilledema, or marked difference, suggests the cerebrum and is, on the whole, in favor of the tumor being on the same side as the greatest swelling when there are other reasons for localizing one in front of the cerebellum.

Intense papilledema may exist *without failure of vision.* When this occurs it is apt to denote the oncoming of atrophy.

Distortion of the visual fields may be present, and color perception is apt to be lost before that of form (p. 600).

In *tumors of the hypophysis* other changes occur (p. 722).

Mental Symptoms.—Mental symptoms, such as irritability, failure of memory, hallucinations, apathy and dementia, may be present; they are especially frequent in lesions of the frontal lobes (p. 614). Hallucinations may have localizing value in locating lesions in certain sensory areas (*infra*).

Vomiting is more frequent in children. It may or may not be accompanied with nausea and has no relation to the taking of food. It often occurs without any warning, and is then known as *projectile vomiting.*

Vertigo may occur with or independently of vomiting. It is most common in tumors in the posterior fossa.

Apoplectiform attacks are due to hemorrhage occurring within or near a tumor, and cause an exacerbation of the symptoms, which may subside in a few days.

Insomnia occurs most frequently in those suffering from syphilitic growths.

Polyuria, with or without glycosuria, may be due to tumor anywhere in the brain. Glycosuria usually indicates one near the fourth ventricle.

Amenorrhea may be an early symptom in women; especially so in hypophyseal growths.

Focal Symptoms.—These are due to either irritation or destruction of the center, or tract, in which the growth is situated, and also to either irritation of neighboring centers, tracts, or cranial nerves, or interference with their function by pressure. If the growth is in a so-called silent region, that is, when there are no centers of known function—as the right temporal lobe in left-handed people—focal symptoms are absent, unless they act upon centers, or tracts, situated near them.

False localizing symptoms may occur, owing to the distortion of the brain, due to the pressure of the growth. Collier has called attention to the following **sources of error:**

1. Local signs appearing *late* in the course of intracranial tumor, when *general signs alone* have preëxisted, are often of false portent.

2. In such cases the local signs have often been due to the presence of independent and later developed vascular lesions, meningitis, hydrocephalus (developed secondarily to tumor, p. 682), which may be a cause of Jacksonian fits; when they occur late in the disease, to local spreading edema of the brain (localized edema in the region of the tumor is frequent), to secondary deposits of new growths and secondary degeneration.

3. The absence of the usually local signs during the early days of illness is in itself an important localizing sign, as it confines the disease to the supratentorial region.

4. True localizing signs, at one time present, may later become concealed or undemonstrable, owing to the development of other signs; and that in cases which come under observation for the first time, late in the disease, diagnosis may be difficult, erroneous or impossible.

Therefore in attempting to localize a growth, attention must be paid to the sequence in which the symptoms have developed—not too much stress being laid upon those which appear late. A *slow growing tumor* which merely compresses the brain may not cause symptoms until it reaches a large size.

The reader is referred to the Chapter on Cerebral Localization, remembering that not only the center, or tract, itself but also neighboring parts are apt to be interfered with and cause symptoms.

Thus a growth in the left third frontal convolution will first cause motor aphasia, and by pressure in the motor centers, cause more or less motor paralysis.

Certain **symptoms peculiar to important regions** may be mentioned:

Frontal Lobes.—Tumor may cause mental symptoms similar to those of paresis. A peculiar feature is a tendency to joke (*Witzelsucht*). Kennedy states that an important symptom is the finding of a retrobulbar neuritis with central scotoma and primary optic atrophy on the side of the lesion and papilledema in the opposite eye.

There may be loss of smell which, if one-sided, is on the side of the tumor. Cerebellar gait has been noted, also perseveration (p. 559). If on the left side, motor aphasia and apraxia may occur. If the third frontal convolution on the left side is involved, motor aphasia results (p. 618). Hemiplegia may develop later.

Motor Region.—If springing from the meninges or cortex primarily, Jacksonian attacks (p. 767) usually occur, followed by motor paralysis. If motor paralysis develop first and Jacksonian attacks some time later, the lesion is probably subcortical. A *slowly developing hemiplegia* (weeks or months) is very suggestive of subcortical tumor in the motor region; cerebral thrombosis may also (very rarely) do this (p. 711) and multiple sclerosis may so develop (p. 744).

Parietal Lobe.—A growth here will cause disturbances of sensation and muscle sense (p. 577), astereognosis and sometimes homonymous hemianopsia. If on the left side, there may be word blindness (*angular gyrus*).

Occipital Region.—Homonymous hemianopsia will be caused (p. 600). Visual hallucinations, such as flashes of light, may occur, usually on the side opposite the growth (p. 768). If on the left side, mind blindness (p. 617) may be present.

Temporal Lobes (See p. 614).

Uncinate Gyrus (p. 614). Tumor here may cause hallucinations of smell and peculiar attacks described on page 768.

Tumors of the Corpus callosum (p. 620). These may cause mental symptoms similar to those caused by disease of the frontal lobes, motor apraxia (p. 617) and hemiplegia, first involving one side, then the other.

The functions of the *corpus striatum, optic thalamus, crus cerebri and tegmentum* are detailed on pages 620-621.

Tumors of the Corpora quadrigemina and Vicinity.—Tumors of the corpora quadrigemina and vicinity (Fig. 140) owing to their connection with the cerebellum cause the cerebellar gait, also ophthalmoplegia, and *loss of associated movement of the eyes upward,* possibly homonymous hemianopsia (Wernicke's papillary inaction sign present, p. 591), dullness of hearing and weakness of the limbs on the contralateral side.

Tumors Springing from Third Ventricle.—Tumors springing from the third ventricle may involve the aqueduct of Sylvius and the superior cerebellar peduncles and cause similar symptoms. If the growth is confined to the ventricle without involvement of aqueduct and peduncles, the symptoms are those of internal hydrocephalus plus the general symptoms of tumor.

Tumor of the Pons.—The symptoms of a lesion of the pons are given on page 622). In addition, tumor may cause nystagmus by involvement of the cerebellar peduncles. If present it is not increased by stimulation (p. 583).

Cerebellum.—The symptoms of disordered function of the cerebellum are given on page 582. Tumors in this region are relatively common.

The *symptoms differ somewhat according to their location*—i. e., in the vermis or lateral lobe. Those situated in either the cerebellopontile angle or so that they involve the cerebellar peduncles also cause cerebellar symptoms. Tumors in this region are especially liable to cause general symptoms (p. 718). If confined to the *vermis* the synergic movements of the trunk, shoulder, and pelvic girdles suffer principally; hence the chief symptoms are disorders of station and gait (p. 582). Nystagmus is usually present. Absence of "past pointing" is a significant symptom (p. 584). Vertigo is a frequent symptom (p. 646).

A symptom termed *cerebellar catalepsy* has been described. It is present if the patient, while reclining on his back, can hold both legs free from the bed for a considerable time without fatigue. Pressure on the aqueduct of Sylvius is apt to occur if the tumor is large, when symptoms of internal hydrocephalus will be present.

Lateral Lobe.—When the lateral lobe is principally involved the symptoms of asynergy (*dysmetria, adiadochokinesis*) are either more marked or present only on the affected side. The cranial nerves from the sixth to the eighth may be involved, but are more apt to be so if the lesion is extracerebellar.

Cerebellopontile Angle.—Tumors of the cerebellopontile angle are usually encapsulated fibromata which spring from either the auditory or trigeminal nerves—usually the former. The first symptoms are due to pressure on a cranial nerve. If the auditory, attacks resembling those of Ménière's disease occur and paralysis of the facial nerve of the supranuclear type is usually associated; if the trigeminal, pain of the neuralgic type associated with diminished sensibility in the distribution of the nerve occurs. Other cranial nerves may become involved and symptoms of pressure on the lateral lobe of the cerebellum and pons be present.

There may be pressure on the pyramidal tract as it passes through the pons, causing spastic paralysis of the arm and leg on the contralateral side. The differential signs between tumors located here and in the cerebellum, cerebellar peduncles, and pons and parietal lobe are given in the table on the following page.

Other Conditions to Be Differentiated from Tumor of the Cerebellum

Tumor of the cerebellum also may have to be distinguished from thrombosis of the superior and inferior cerebellar arteries.

THROMBOSIS OF THE SUPERIOR AND INFERIOR CEREBELLAR ARTERIES.

Thrombosis of both the superior and inferior cerebellar arteries cause symptoms of cerebellar disease. They would, however, appear suddenly (See p. 712).

Tumor of the Gasserian Ganglion.

Tumor of the Gasserian ganglion causes intense pain and diminished or lost sensation in the fifth nerve distribution. Cerebellar symptoms and paralysis of other cranial nerves will be absent.

Tumor of the Pituitary Body or Hypophysis.

Tumor of the pituitary body or hypophysis and its vicinity usually makes pressure upon the optic chiasm, and hence *bitemporal hemianopsia*

	Cerebellum Lateral Lobe	Cerebellopontile Angle	Pons and Middle Cerebellar Peduncles	Superior Cerebellar Peduncles. Region of Aqueduct of Sylvius and Corpora quadrigemina	Parietal Lobe
General Symptoms, Papilledema, etc.	Intense	May or may not be present	Often absent	Often absent	Often absent
Vertigo	Subjective rotation of self from side of lesion	Subjective rotation of self to side of lesion; may be paroxysmal	Not definite	Indefinite	Absent
Incoördination	Symptoms (p.582) marked on side of lesion; gait disturbed	Not so marked, but usually present	Not marked	May be marked on side of lesion	Present; also astereognosis
Nystagmus (p. 583)	Spontaneous; may be absent. Past pointing absent.	Spontaneous. If 8th nerve affected, is not increased by vestibular stimulation. Past pointing absent	Absent or may be weak after vestibular nerve is stimulated. Past pointing present	Weak and not increased by stimulation. Past pointing may be absent	Absent
Cranial Nerve Paralysis	Absent if lesion confined to cerebellum	Present especially side of lesion 5th, 7th, 8th. Marked vertigo and tinnitus. Other nerves below 8th may be affected	Usually present side of lesion, especially 5th, 6th, 7th. Loss of conjugate lateral movement of eyes to side of lesion	3rd nerve, possibly 4th. Loss of associated upward movement of eyes; slight deafness	May be homonymous lateral hemianopsia
Paralysis Motor Tracts	Absent. Reflexes often diminished unless internal hydrocephalus occurs	May be spastic paralysis opposite side of lesion. Reflexes increased. Babinski present	Spastic paralysis opposite side, sometimes both sides	May be spastic paralysis; opposite side if crus involved, or both sides may be weak with increased reflexes, etc.	May be spastic paralysis, hemiplegic type. Possibly Jacksonian convulsions. If on left side word blindness
Sensation	Not disturbed	Not disturbed	May be hemianesthesia; often face on side of lesion, arm and leg on opposite	May be disturbed from pressure on tegmentum	Not disturbed

(p. 600) is one of the earliest symptoms. Frequently this is first manifested in the color fields. In a small proportion of cases lateral homonymous hemianopsia has been noted. Total blindness, first in one eye, sooner or later occurs due to *primary optic atrophy* (p. 599).

X-ray examination frequently reveals enlargement and deformity of the sella turcica. Headache is often severe, and epileptiform convulsions may occur. Menstrual disturbances have localizing value (*infra*). The symptoms referable to the hypophysis itself are due to disturbance of the function of the anterior portion and depend upon whether there is hypersecretion or hyposecretion; if the former occur in one still in the growing period of life, gigantism or excessive growth, shown by abnormal height and size of bones, occurs; if it occurs in one who has reached his full stature, acromegaly results (p. 519). If hypersecretion is not caused by the growth these symptoms will not occur. When the latter or dyspituitarism is the case the most striking symptoms are those known as infantilism, or the so-called *dystrophia adiposa genitalis of Froehlich.* These consist of an excessive development of adipose tissue (this may not occur in all cases), failure or absence of sexual powers and feelings (amenorrhea in females), lack of development of the sexual organs and lack of growth of pubic hair, and in some cases an increased tolerance of carbohydrates. Symptoms of hypersecretion may occur first and later become those of hyposecretion.

Conditions to Be Differentiated from Tumor of the Brain

Tumor may have to be distinguished from:

Brain abscess
Serous meningitis
Chronic internal hydrocephalus
Tubercular meningitis
Uremia
Chronic anemia
Disease of accessory nasal sinuses
Thrombosis in cerebral vessels
Cerebral apoplexy
Multiple sclerosis
Hysteria
Paresis.

BRAIN ABSCESS.

Brain abscess would only be suspected when the symptoms followed one of the causes of such trouble (p. 716). Leukocytosis and fever with slow pulse would be in favor of abscess.

Serous Meningitis.

Serous meningitis may be hard to distinguish. Cases of supposed tumor which recover (pseudotumor) are often due to it. If the symptoms improve after lumbar puncture serous meningitis must be suspected. It must be remembered that this is a dangerous procedure in brain tumor, especially when it is situated in the posterior fossa. If it is done the fluid must be withdrawn very slowly.

Chronic Internal Hydrocephalus.

Hydrocephalus frequently occurs in association with tumor (p. 682). If due to other causes the paralysis is bilateral; this rarely occurs in tumor except in those in the pons. X-ray examination may show thinning of the cranial bones.

Tubercular Meningitis.

In tubercular meningitis there may be a localized collection of tubercle which, to all intents and purposes, is a tumor which may cause focal symptoms. This may be associated with a general tubercular meningitis. If so, the general symptoms of meningitis and peculiar characteristics of the cerebrospinal fluid (p. 56) will show the true condition.

Uremia.

Uremia may cause a condition in the eye grounds simulating papilledema. Careful examination should reveal the true cause.

Chronic Anemia—Disease of the Sinuses.

The same may be said of anemia and disease of the sinuses.

Thrombosis in Cerebral Vessels.

A slowly forming thrombosis in cerebral vessels may simulate the gradually developing hemiplegia without general symptoms that may be caused by tumor in the motor region. The distinction may be difficult. The former, however, occurs comparatively rarely. Jacksonian attacks usually occur sooner or later in tumor. Disease of the heart (myocardial) and arterial degeneration would always be present in the former.

Cerebral Apoplexy.

Apoplectiform attacks may occur in the course of tumor (p. 710). They lead to sudden exacerbation of the symptoms which have preceded the attack and after it passes off they will become nearly, if not entirely, as they were before.

Multiple Sclerosis.

A slowly developing hemiplegia may also occur in multiple sclerosis (See p. 688).

HYSTERIA.

Hysteria may occur in association with tumor. Careful examination will show that symptoms of organic disease, as outlined in the symptomatology of tumor, are present.

PARESIS.

Tumors of the frontal lobe may simulate paresis. The physical symptoms of the latter are different, viz., tremor of hands, tongue, face; Argyll-Robertson pupil, peculiar speech (p. 752).

2. Amaurotic Family Idiocy

Characteristic Features.—Amaurotic family idiocy, or Tay-Sachs' disease, is characterized by failing mental functions, usually appearing when the child is between six months and a year old, but which may appear later; weakness and paralysis of the limbs, which may be either flaccid or spastic; increased, decreased or absent tendon reflexes; failure of vision progressing to blindness, due to a peculiar form of atrophy of the optic nerve, and death in one to two years.

Occurrence.—The disease is most common in Jews and is hereditary.

Conditions to Be Differentiated from Amaurotic Family Idiocy

The following conditions must be distinguished from this disease:

Amyotonia congenita
Hereditary amaurotic ataxic paraplegia.

AMYOTONIA CONGENITA.

In amyotonia congenita blindness does not occur; the limbs are extremely flaccid; there is absence of mental deterioration.

HEREDITARY AMAUROTIC ATAXIC PARAPLEGIA.

The symptoms of hereditary amaurotic ataxic paraplegia are described on page 698.

H. Focal and Diffuse Diseases of the Spinal Cord

Affections of the Blood Vessels

The main arteries of the spinal cord are three in number; they arise from vertebrals.

The anterior spinal artery passes down the anterior surface of the cord in front of the anterior median fissure. The two posterior spinal arteries pass down the posterior surface on each side near the point of entrance of the posterior nerve roots. They all anastomose freely with each other.

(*a*) *Embolism and Thrombosis*

Embolism and thrombosis may occur in these vessels (pp. 729, 748).

(*b*) *Hemorrhage*

Hemorrhage is almost always due to traumatism, and may occur either into the meninges and vertebral canal or cord.

1. Spinal Meningeal Hemorrhage

Occurrence and Causes.—Spinal meningeal hemorrhage, or *hematorrhachis*, occurs in newly born children and adults, and may be inside or outside the dura. In the latter the usual causes are injuries, with or without fracture of the vertebra. Severe convulsions, muscular exertion, purpura, infectious fevers, and bursting of an aortic or vertebral aneurism may be causes.

Symptoms.—The symptoms consist of pain in the back, root pains and muscular twitchings due to irritation of the nerve roots, and if large enough to cause pressure on the cord, more or less motor and sensory paralysis. Lumbar puncture will show the fluid to be bloody.

If outside the dura the blood may gravitate downward at first; then as it increases in amount, the symptoms creep upward as in ascending paralysis.

Diagnosis.—The diagnosis depends upon the *sudden* onset of the symptoms following one of the causes mentioned. It must be distinguished from hemorrhage into the cord.

HEMORRHAGE INTO THE CORD.

In this pain and symptoms of root irritation (unless there is fracture or dislocation of the vertebra) are much less and the paralytic symptoms develop more suddenly and are more severe.

2. Hematomyelia

(*Hemorrhage into the Cord*)

Etiology.—This condition is almost always due to a fall or blow upon the back or the buttocks. Fracture and dislocation of the vertebra (See p. 734) may be associated. It may occur secondarily to myelitis or spinal tumor. It may follow excessive sexual indulgence. Arterial degeneration, purpura, and convulsions are rare causes.

The gray matter of the lower cervical region and conus are the most common seats of the hemorrhage. It is usually single, but multiple hemorrhages may occur.

Symptoms.—The symptoms consist of the *sudden* appearance of numbness and weakness of the limbs rapidly developing into complete paralysis. The location of the symptoms depends upon the location of the hemorrhage; this can be determined by reference to page 625. After the onset the symptoms usually improve unless death occur, leaving a more or less severe permanent paralysis.

Conditions to Be Distinguished from Hematomyelia

This condition must be distinguished from:

Spinal hemorrhage
Fracture and dislocation of the vertebra
Acute myelitis
Syringomyelia.

SPINAL HEMORRHAGE.

The distinguishing points of spinal hemorrhage are given on page 727.

FRACTURE AND DISLOCATION OF THE VERTEBRA.

Fracture and dislocation of the vertebra are frequently accompanied by hemorrhage. The resulting kyphosis, and if that is absent, x-ray examination, will reveal the true condition. The sudden development of paralysis following a back injury or fall, if fracture can be excluded, is due to hemorrhage.

ACUTE MYELITIS.

Acute myelitis does not develop immediately after a traumatism, although it may within a day or two. It is usually due to other causes (p. 729).

SYRINGOMYELIA.

The symptom complex following hemorrhage, especially if in the cervical region, may resemble that of syringomyelia. This is due to the hemorrhage occurring in the gray matter and causing *dissociation of sensation* (p. 577). Syringomyelia is, however, a disease of gradual onset.

(*c*) *Caisson Disease*

Caisson disease has been discussed on page 215.

(*d*) *Inflammation of the Spinal Cord*

(*Myelitis*)

This may be acute, subacute or chronic.

If the gray matter alone is affected, it is termed *poliomyelitis;* if a small vertical extent of the entire surface of the cord (several segments), *transverse myelitis;* a large extent of gray and white matter, *diffuse myelitis;* if in scattered areas, *disseminated myelitis;* if following hemorrhage, *hemorrhagic myelitis;* if due to compression by bone disease, fracture of the vertebra, or tumor, *compression myelitis.*

1. Acute Myelitis

Occurrence.—Most cases occur between the ages of ten and forty years.

Causes.—The causes are toxic agents, especially the acute infectious fevers, infection during the puerperium, and syphilis; more rarely, the metallic poisons; falls or blows upon the back, which may also cause primarily either hematomyelia or fracture and dislocation of the vertebra, the myelitis being secondary (*vide*); or the inflammation may be the primary and only lesion; extension of inflammation from neighboring parts, as the meninges or vertebra; exposure to cold and wet.

Thrombosis or embolism in the vessels of the cord, with consequent softening, causes symptoms that often cannot be distinguished from those of myelitis. This is known as *myelomalacia.* It occurs usually in old people or is due to syphilis (See also p. 748).

The most common type, exclusive of poliomyelitis (See p. 188), is the transverse and the dorsal region, the most common location affected.

If due to trauma without coexisting hemorrhage or fracture of the vertebra, the symptoms may not develop for several days after.

Symptoms.—The first symptom noticed is usually numbness of the legs, soon followed by weakness, and in a day or two, or even less time, the paralysis of motion and sensation in parts below the level of the lesion is complete.

The location of the lesion must be determined by methods detailed under spinal localization (p. 625).

In addition, at first there is retention of urine followed by incontinence, or there may be incontinence from the beginning (p. 592). If confined to the cord there is no pain; if the meninges are also affected there is a feeling of constriction of the body at the level of the lesion (girdle sensation), and often a zone of hyperesthesia just above it. Bedsores, especially over the sacrum, buttock and heels, usually soon develop. These may be very severe, causing sometimes rapid sloughing of an entire buttock.

If in the cervical region, paralysis of the cervical sympathetic may also occur.

Fever is often present. If the patient survive, more or less improvement gradually occurs, sensation usually returning first, and if the lesion is in the cervical or dorsal region, a more or less spastic or ataxic paraplegia results (chronic myelitis).

The usual distribution of symptoms, according to the region involved, is shown in the table of Prince:

	Lumbar Myelitis	Dorsal Myelitis	Cervical Myelitis
Paralysis	Paraplegia	1. Dorsal, abdominal, and intercostal muscles according to height of lesion. 2. Legs	Neck muscles, diaphragm, arms, trunk, and legs
Sensation	Pains in legs, or girdle pains around loins; hyperesthetic zone around loins; anesthesia of legs, complete or uneven distribution	Girdle pain and hyperesthetic zone between ensiform cartilage and pubes	Hyperesthesia and pains in certain nerve distributions of arms; below this anesthesia of arms, body, and legs
Atrophy	Of legs	Of dorsal and abdominal (and intercostal muscles, not subject to examination) corresponding to height of lesion; sometimes mild and slow of legs	Atrophy of neck muscles (rare) or more commonly of arms
Electrical Reaction.	De R in atrophied muscles or in mild cases quantitative diminution	De R in dorsal and abdominal muscles; slight quantitative changes only in legs when wasted	De R in atrophied muscles
Bladder	Incontinence from paralysis of sphincter	Retention, or intermittent incontinence from reflex actions; later from overflow. Cystitis common	Same as in dorsal myelitis
Bowels	Incontinence from paralysis of sphincter, disguised by constipation	Involuntary evacuation from reflex spasm, or constipation	Same as in dorsal myelitis
Reflexes, superficial.	Lost	Temporary loss, then rapid increase	Same as in dorsal myelitis
Reflexes, deep	Lost	Temporary loss, then slow increase	Same as in dorsal myelitis
Priapism	Absent	Often present	Often present

From the original focus the inflammation may extend either up or down the cord (diffuse myelitis) causing symptoms of an ascending or descending paralysis.

Disseminated myelitis is due usually to the poison of an infectious disease or septic process, and may be associated with encephalitis. The symptoms resemble those of multiple sclerosis plus acute onset and fever.

Conditions to Be Distinguished from Acute Myelitis

The acute disease may have to be distinguished from:

Hematomyelia
Fracture and dislocation of the vertebra
Myelomalacia (p. 729)
Anterior poliomyelitis
Landry's disease
Multiple neuritis
Lesions of the cauda equina
Hysterical paraplegia.

HEMATOMYELIA.

In hematomyelia the development of the symptoms is much more rapid—often almost instantaneous, and almost invariably follows trauma.

FRACTURE AND DISLOCATION OF THE VERTEBRA.

Fracture or dislocation of the vertebra can usually be discovered by examination; if the kyphosis is not present, x-ray examination will show them if they exist.

MYELOMALACIA.

Myelomalacia often cannot be distinguished. It occurs in old people in whom none of the etiological factors mentioned have been present; it is due in these cases to arterial degeneration. When it occurs in the young, syphilis is the cause. Fever in such cases is usually absent.

ANTERIOR POLIOMYELITIS.

In acute anterior poliomyelitis there is absence of sensory paralysis; the sphincters are not usually involved; bedsores do not occur.

LANDRY'S DISEASE.

Landry's disease may be due to myelitis (See p. 696).

The development and extension of the symptoms are rapid, and in the typical condition sensory symptoms are absent.

MULTIPLE NEURITIS.

In multiple neuritis there is usually pain and tenderness over the affected nerve trunks. The sensory paralysis, if present, is not symmetrical; the sphincters are rarely involved. The two conditions may sometimes coexist.

LESIONS OF THE CAUDA EQUINA.

Lesions of the cauda equina may resemble myelitis in the lumbosacral region. The differential points are given on page 629.

HYSTERICAL PARAPLEGIA.

Paraplegia due to hysteria may develop suddenly, but sphincter paralysis, the Babinski reflex, and bedsores would not occur. Other stigmata *of hysteria* are present (pp. 777, 785).

TRANSVERSE, DISSEMINATED AND DIFFUSE MYELITIS may have to be distinguished from each other. The differential points have been given in the description of the symptoms.

2. Chronic Myelitis

Etiology.—Chronic myelitis may follow an acute attack of myelitis or meningitis. As a primary disease it is very rare but it is said may be due to syphilis, exposure, alcoholism, infectious diseases, falls or blows upon the back or buttocks.

This title is most usually applied to the resulting permanent symptoms of a previous acute attack (p. 729). When it occurs primarily, the symptoms develop slowly, sensory paralysis is not so complete and total loss of power either never occurs, or results only after a long time. If the meninges are also affected girdle pain (p. 567) and pains in the legs are complained of. Transverse myelitis in the dorsal region is the usual form present.

Symptoms.—The symptoms characteristic of affection of different parts of the cord are given on page 625. They are often not so well marked in the chronic disease.

Conditions to Be Differentiated from Chronic Myelitis

The diagnosis is easy if preceded by an acute attack; when it does not it must be distinguished from:

Primary lateral sclerosis
Compression myelitis
Spinal tumor
Spinal syphilis (Erb type)
Combined sclerosis
Amyotrophic lateral sclerosis
Spinal pachymeningitis
Senile paraplegia
Multiple sclerosis.

PRIMARY LATERAL SCLEROSIS.

In primary lateral sclerosis there is usually no involvement of the sphincters; sensory symptoms are absent. In some cases the distinction may be difficult.

COMPRESSION MYELITIS.

Compression myelitis (*infra*) is distinguished by finding disease of the vertebra or other cause of pressure upon the cord. Sensory symptoms, especially pain of the root type, are usually more intense than would occur in myelitis. X-ray examination may help.

SPINAL TUMOR.

Tumor of the spinal cord, if outside the cord, causes pain of the root type and loss of sensation of the segmental type (p. 567). The cord symptoms at first are apt to be asymmetrical.

If intraspinal, pain may be absent, but the symptoms develop asymmetrically. Dissociation of sensation may be present, which is not so in myelitis.

SPINAL SYPHILIS.

In spinal syphilis of the Erb type sensory symptoms, except slight pain, are usually absent. Spasticity is often pronounced when the patient walks, but disappears when he is at rest.

COMBINED SCLEROSIS.

Combined sclerosis due to anemia or other cause will be distinguished by the peculiar development of the symptoms and the presence of the cause (p. 702).

AMYOTROPHIC LATERAL SCLEROSIS.

In amyotrophic lateral sclerosis atrophy begins in the small muscles of the hands. Fibrillary tremors are present; sensory symptoms are absent.

SPINAL PACHYMENINGITIS.

In spinal pachymeningitis pain of root type is severe; there are apt to be muscular twitchings which usually precede the development of paraplegia. When this occurs it depends on pressure and may be slight.

SENILE PARAPLEGIA.

Senile paraplegia occurs in old people with arteriosclerosis. The gait is peculiar, consisting of short, shuffling steps and is not typically spastic. Sensory symptoms are usually absent; mental deterioration may be present; the Babinski reflex is often absent.

MULTIPLE SCLEROSIS.

Typical cases of multiple sclerosis are not likely to be mistaken; atypical ones may be. Usually nystagmus, atrophy of the optic nerves or other symptoms which occur in multiple sclerosis—and not in myelitis—will be found.

3. Compression Myelitis

Compression of the spinal cord (compression myelitis) may be due to caries of the vertebra, either tubercular or syphilitic, tumor springing from the vertebra and membranes (p. 734), aneurism of the abdominal or thoracic aorta, meningeal cyst, spinal pachymeningitis of Charcot and Joffroy (p. 683), and spondylitis.

Symptoms.—The early symptoms consist of more or less severe pain of the root type, twitchings of the muscles, spinal rigidity and tenderness, possibly kyphosis.

Those referable to the cord are due to gradual and continuous pressure upon the cord. Motor symptoms usually appear before sensory symptoms and are of the spastic paraplegic type.

Diagnosis.—The diagnosis depends upon finding one of the causes above mentioned. X-ray examination will often assist.

4. Senile Paraplegia

Occurrence.—Senile paraplegia occurs in old people with arteriosclerosis.

Symptoms.—It is characterized by sensations of numbness and characteristic gait. This consists of very short shuffling steps, the feet often never leaving the ground. The limbs, however, are not spastic, and while the knee jerks may be increased the Babinski reflex is usually absent.

If there is pain and tenderness of the nerve trunks, *senile neuritis* is also present (See p. 673).

5. Injuries of the Spinal Cord Due to Fractured or Dislocated Vertebra

Causes.—Injuries of the spinal cord due to fractured or dislocated vertebra are usually due to falls or blows upon the back or head or to forcible bending.

Symptoms.—The symptoms may be due to either pressure on or destruction or division of the cord. There is usually kyphosis at the seat of the lesion; if not, x-ray examination will locate it. If evidence of injury of the vertebrae is absent, similar symptoms following immediately the causes mentioned are due to hemorrhage (p. 727); if after a lapse of from a few hours to a few days more, to myelitis (p. 729).

If after a day or two there is flaccid paralysis of the legs and absent knee jerks, the cord has probably been divided. Of course this only applies to lesions above the lumbar enlargement.

If the cord is not divided, symptoms similar to those of transverse myelitis develop, the location of the process being determined by the rules laid down under spinal localization (p. 625).

(*e*) *Tumors of the Spinal Cord*

Occurrence.—Tumors of the spinal cord are not as frequent as tumors of the brain. They are most common in middle life. When they occur in old people they are usually malignant and due to metastasis. They are sometimes multiple and may occur thus in von Recklinghausen's disease (p. 677).

Etiology.—*Injury to the back* may be an exciting cause, especially of sarcomata and cysts. The symptoms of the latter resemble those of neoplasm so closely that they will be described here. They may be parasitic or due to *circumscribed serous spinal meningitis.*

The classification of Bruns is a convenient one:

I. Tumors which arising in its envelopes, secondarily affect the spinal cord.

(*a*) Vertebral tumors arising from the spinal column or the soft tissues immediately surrounding it.

(*b*) Intravertebral tumors, which may be divided into two classes, in accordance with their relation to the dura mater.

(1) Extradural tumors originating in the periosteum of the vertebra, the outer layer of the dura mater, or the fatty areolar tissue of the epidural space.

(2) Intradural tumors originating from the inner layers of the dura, the arachnoid, the ligamentum denticulatum, the spinal roots or the pia mater.

II. Intramedullary tumors of intrinsic spinal origin.

Those arising from the vertebra are usually malignant, either carcinoma or sarcoma and are due to metastasis as a rule. Myelomata may occur. Benign growths, as osteomata, exostosis, chondromata, etc., occur rarely.

Extradural tumors, in the order of frequency, are sarcomata, lipomata, fibromata, myxomata and chondromata.

Intradural tumors may be either diffuse or localized; sarcomata, primary or metastatic; endotheliomata, cylindromata, fibromata and lymphangiomata; fibromyxomata and fibrosarcoma are found in connection with the nerve roots; cysts occur within the dura. These growths are found most frequently in the lateral and posterolateral surfaces of the cord.

Intramedullary growths comprise gliomata, sarcomata, angiosarcomata, gummata, and tubercle.

Glioma usually occurs about the central canal and then may cause the symptoms of syringomyelia (p. 738).

i. Extramedullary Growths

Causes.—The symptoms of extramedullary growths are due to irritation of the nerve roots—especially the posterior, and pressure on the cord.

Symptoms.—The first symptom is almost invariably pain, with sometimes hyperesthesia in the course of the roots irritated. If the anterior roots are involved, twitchings and involuntary clonic contractions of the muscles supplied by them may occur. Later, in addition to pain there is usually either loss or diminished sensibility in the distribution of the affected roots.

As pressure is made on the cord, motor and sensory paralysis and involvement of the sphincters gradually develop. At first these symptoms may be unilateral, and the Brown-Séquard symptom complex result (p. 631).

Diagnosis.—Methods of determining the location of the tumor are given under spinal localization (p. 625).

ii. Intramedullary Growths

The symptoms of intramedullary growths may be those of a slowly developing myelitis (p. 731). The Brown-Séquard syndrome is likely to develop first.

If about the central canal, the symptoms are those of syringomyelia. Pain is either slight or not present until the periphery is reached.

iii. Circumscribed Spinal Serous Meningitis

The symptoms are similar to those of tumor. Sensory symptoms (root pain and hyperesthesia followed by anesthesia) may persist for some time before pressure symptoms develop. A peculiar feature is that the symptoms may vary from time to time, according as the pressure of the fluid increases or diminishes (p. 685).

Conditions to Be Differentiated from Tumor of the Spinal Cord

Tumor of the spinal cord must be distinguished from:

Vertebral caries
Localized serous cysts or circumscribed spinal serous meningitis
Transverse myelitis
Syphilitic meningitis
Spinal pachymeningitis
Syringomyelia
Tabes
Disease of cauda equina
Neuralgia.

VERTEBRAL CARIES.

In vertebral caries due to tuberculosis the root pain is not so great, but the tenderness of the vertebra on pressure and jarring is greater. Tuberculosis may be found elsewhere.

It occurs most commonly in the young; tumor does not. Kyphosis is usually pronounced. In doubtful cases x-ray examination must be made.

LOCALIZED SEROUS CYSTS.

The symptoms of localized serous cysts have been discussed (*supra*).

TRANSVERSE MYELITIS.

In transverse myelitis of acute origin the rapid development of the symptoms excludes tumor. In the chronic form they develop more symmetrically, and root pain and anesthesia do not precede their development.

The cerebrospinal fluid in tumor often has a greenish or yellowish tint, and gives a positive globulin reaction, but the cells are not much, if any, increased in number. If the tumor block up the vertebral canal, it will flow very slowly—possibly not at all.

Intramedullary growths may be difficult, but the appearance of symptoms on one side some time before the other is affected is in favor of tumor.

SYPHILITIC MENINGITIS.

In syphilitic meningitis the number of lymphocytes in the cerebrospinal fluid will be greatly increased and the Wassermann reaction may be, but is not invariably present. The symptoms will probably yield to antisyphilitic medication.

Spinal Pachymeningitis.

The symptoms of pachymeningitis usually develop symmetrically. It occurs in old people (p. 683).

Syringomyelia.

In syringomyelia peculiar trophic symptoms, such as scoliosis, arthropathies, painless ulcerations and suppuration, usually develop. Intramedullary tumor is rare as compared with syringomyelia.

Tabes.

The pain may resemble somewhat that of tabes, but careful examination for the usual symptoms of that disease will make the diagnosis clear.

Disease of Cauda equina.

The differential diagnosis of spinal lesions and those of the cauda equina has been given on page 629.

Neuralgia.

Pain of the character peculiar to spinal tumor and other conditions irritating posterior nerve roots are frequently for a long time treated as neuralgia—especially the *sciatic nerves*. All such cases should be most carefully examined (See pp. 567, 575, 664).

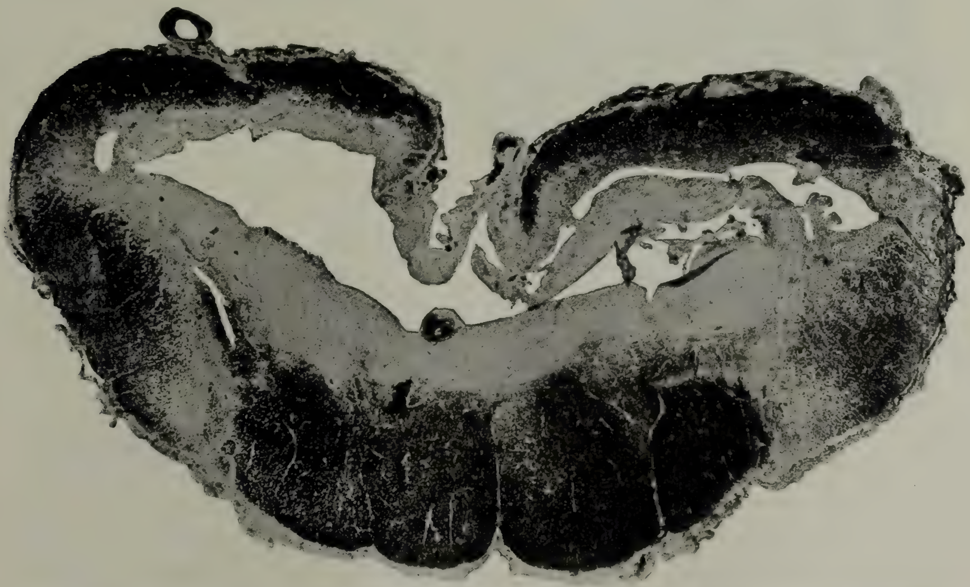

Fig. 178.—Syringomyelia of the Cervical Enlargement. The White Substance Is Stained Black by Weigert's Stain. The Entire Gray Substance Is Changed, Destroyed, or Permeated with Cavities. Even in the Posterior Horn, Only a Part of Its Contour Is Normally Retained.

The nature of the tumor is difficult to determine; in the young it may be tuberculoma; in middle life it is most probably either sarcoma or glioma. Gummata may be surmised by the history and results of serological tests.

(*f*) *Syringomyelia*

Etiology.—Syringomyelia is the term applied to a symptom complex produced by either an acquired enlargement of the central canal of the spinal cord or by the formation of new canals in the gray matter of the cord. This formation may extend into the medulla, the symptoms so caused being known as syringobulbia.

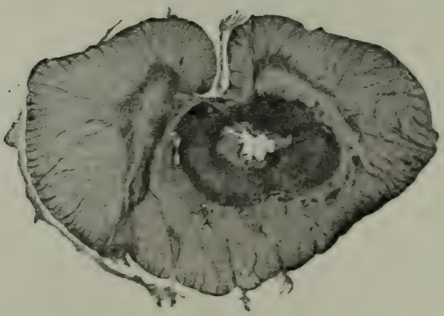

Fig. 179.—Transverse Section through the Dorsal Portion of the Spinal Cord. Tumorlike Accumulations of Glia Around a Central Cavity (Gliosis).

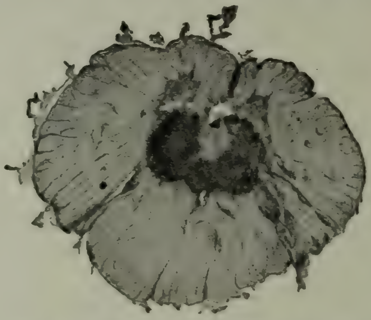

Fig. 180.—Transverse Section through the Dorsal Portion of the Spinal Cord (from the same case as Fig. preceding). There Is No Central Cavity, but Only Rarefaction of the Greatly Proliferated Central Glia Masses.

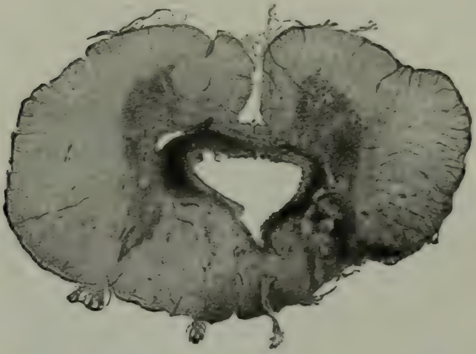

Fig. 181.—Transverse Section of a Dorsal Portion of the Lumbar Cord (from the same case as Fig. 177). A Larger Central Cavity with a Marked Glia Ring.

Various causes bring about the development of this cavity, viz.: trauma, in which it is probably due to hemorrhage (p. 727), followed by the formation of cavities and cysts; inflammatory processes following the infectious diseases; arterial degeneration due to unknown cause and followed by thromboses and softening; congenital anomalies of development; the extensive formation of glia cells (gliosis), which may amount to actual tumor formation (gliomatosis, gliomata, p. 718) (Figs. 178-181). The last is the most common condition.

Occurrence.—The disease is more common in males and usually develops between the ages of twenty and thirty. It may affect several members of the same family.

Symptoms.—The location of the pathological process explains the symptoms. As the gray matter is more or less destroyed, the fibers conducting pain and temperature sense are cut off (p. 556) and the white matter pressed upon.

The symptom first noted is usually atrophy of the small muscles of the hands and claw-hand deformity (Fig. 164). After this the muscles of the arms become involved.

Fibrillary tremors are present; usually the legs are spastic with increased tendon reflexes and the Babinski reflex. In some cases the knee jerks are absent, or there may be increase on one side and loss on the other.

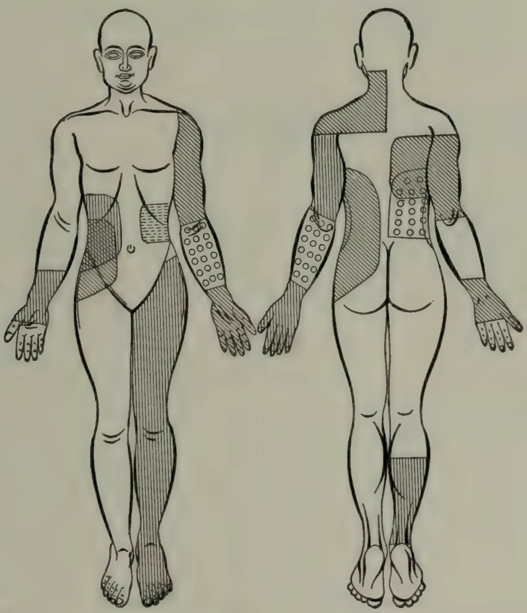

Fig. 182.—Sensory Chart of Patient, Showing Areas of ▧ *Thermo-anesthesia,* ▥ *Analgesia,* ▩ *Thermo-anesthesia and Analgesia,* ▤ *Tactile Anesthesia,* and Areas in Which the Patient's Answer to Tests of Temperature Showed Reversal ⁘ *Cold-Hot, Hot-Cold.* (After Potts' "Mental and Nervous Diseases," published by Lea and Febiger, Philadelphia.)

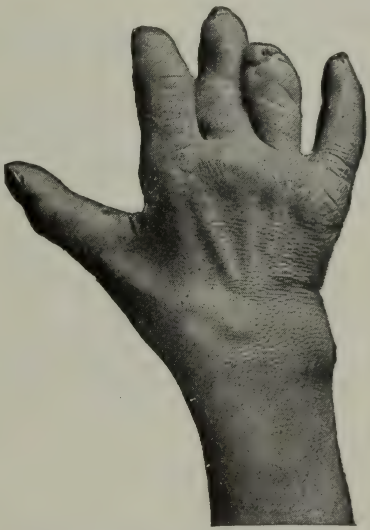

Fig. 183.—Syringomyelia of the Cervical Cord. Deformity of the Hand, Atrophy of the Interossei and of the Adductor Pollicis. (After Strümpell.)

The sensory changes are characteristic, there being always some areas in which there is dissociation of sensation (p. 577). There may be others in which there is only loss of temperature sense, while in others all forms may be lost (Fig. 182).

Pain and paresthesia referred to different parts of the body may be complained of; scoliosis develops sooner or later; various trophic disturbances, as arthropathy—especially of the shoulder—fragility of the bones, hypertrophy of the bones, facial hemiatrophy (p. 640) and ulceration of the skin may develop. Painless swelling and suppuration of the fingers with consequent necrosis and (sometimes) loss of the bones (painless felons) are not uncommon (Fig. 183). This condition has been termed *Morvan's disease.*

Paralysis of the cervical sympathetic may occur, and vasomotor symptoms, as cyanosis, coldness of the extremities and excessive sweating, are common.

In syringobulbia there is involvement of the bulbar nuclei and paralysis of the vocal cords, paralysis of the tongue with atrophy (sometimes unilateral) and other bulbar symptoms may be present. More rarely there may be also sensory loss in the distribution of the fifth nerve. If the condition originates in the lumbar region muscular atrophy will appear first in the legs.

Conditions to Be Differentiated from Syringomyelia

Syringomyelia must be distinguished from:

Progressive spinal muscular atrophy
Amyotrophic lateral sclerosis
Chronic bulbar palsy
Cervical pachymeningitis
Vertebral caries
Tabes dorsalis
Spinal tumor
Muscular dystrophy
Multiple sclerosis
Myelitis
Hematomyelia
Leprosy
Raynaud's disease
Scleroderma
Arteriosclerosis.

PROGRESSIVE SPINAL MUSCULAR ATROPHY.

In progressive spinal muscular atrophy sensory symptoms are absent.

AMYOTROPHIC LATERAL SCLEROSIS.

The same may be said of amyotrophic lateral sclerosis.

CHRONIC BULBAR PALSY.

Syringobulbia may be mistaken for chronic bulbar palsy. In the latter the symptoms develop symmetrically; sensory symptoms are absent. It usually affects older people.

Cervical Pachymeningitis.

In cervical pachymeningitis pain, which is of the root type, is usually greater; dissociation of sensation may occur, but not so commonly as in syringomyelia; there is often rigidity and tenderness of the neck.

Vertebral Caries.

Vertebral caries can be recognized by the presence of kyphosis, tenderness on jarring, pressure, and x-ray examination.

Tabes dorsalis.

Syringomyelia in which arthropathies develop and the knee jerks are absent may be confounded with tabes dorsalis. In tabes pain is usually more pronounced, Argyll-Robertson pupil is commonly found and optic atrophy is often present. Serologic examination is important (p. 754).

Spinal Tumor.

Spinal tumor developing in the gray matter may be impossible to differentiate; if extraspinal the pain is more pronounced. Trophic symptoms do not develop.

Muscular Dystrophy.

In muscular dystrophy fibrillary tremors and sensory symptoms are absent. The disease usually appears in childhood.

Multiple Sclerosis.

Typical multiple sclerosis is not liable to be mistaken for syringomyelia; cases in which muscular atrophy and sensory loss occur may lead to some confusion. In it, however, trophic symptoms never occur and some of the characteristic symptoms, as atrophy of the optic nerve on the temporal side, diplopia, or tremor, will be found.

Myelitis.

The symmetrical distribution of the symptoms in transverse myelitis is not characteristic of syringomyelia. Dissociation of sensation is rare; there are more pronounced disturbances of the functions of the bladder and rectum; muscular atrophy does not occur unless the cord is affected in either the cervical or lumbar regions.

Central myelitis in which the gray matter alone is involved may be difficult if not impossible to distinguish.

Hematomyelia.

Hematomyelia always has an acute onset. The resulting permanent condition may simulate syringomyelia.

Leprosy.

Leprosy affecting the peripheral nerves may be mistaken. In this disease nodular thickening of the peripheral nerves may be felt. The facial nerve is often the first to be affected, while other cranial nerves escape. The bacillus of leprosy can usually be found in the nasal mucus. Spastic paraplegia does not occur and the skin will probably show some of the evidences of the disease.

Raynaud's Disease.

Cases in which skin ulcerations occur may be mistaken for Raynaud's disease. In this, sensory changes, muscular atrophy and spastic paraplegia are absent. At first there is a history of periodical attacks of local syncope and asphyxia.

Scleroderma.

Scleroderma in which sclerodactylia occurs may be similarly mistaken. Symptoms pointing to the spinal cord are, however, absent.

Arteriosclerosis.

Arteriosclerosis causing so-called senile neuritis (p. 673) may cause symptoms simulating those of syringomyelia. The marked muscular atrophy, dissociation of sensation and paraplegia are absent.

I. Diffuse Diseases Affecting the Brain and Spinal Cord

(*a*) *Multiple Sclerosis*

Multiple sclerosis, also termed insular sclerosis, disseminated sclerosis, and multiple cerebrospinal sclerosis, is a comparatively rare chronic disease in which there are areas of sclerosis scattered through the brain and cord. It differs from the ordinary forms of sclerosis in that the myelin sheath is affected first and disappears, the nerve fiber remaining intact for sometime afterward.

Occurrence.—Most cases develop between the age of twenty and thirty, although children may be affected.

Etiology.—Some cases seem to follow either exposure, traumatism, emotional shock or depression, infectious diseases, chronic metallic poisoning, or overwork. A defect in the development of the nervous system may be a predisposing cause. A hereditary disease (Batten, Brain, 1914, p. 341), developing in early childhood, in which the symptoms resemble those of multiple sclerosis, has been described (aplasia axialis extracorticalis congenita).

Symptoms.—The symptoms may develop more or less acutely, but usually the onset is slow and the course progressive. Ophthalmoplegia and diplopia may be early symptoms. Usually spastic paraplegia is first complained of, or the gait may resemble that of ataxic paraplegia. The deep reflexes are very much increased and the Babinski reflex is usually present, but the abdominal reflex is often absent.

A coarse tremor or ataxia only present when the arms are moved, as in the finger to nose test; either a scanning (singsong) or slurring speech; nystagmus either spontaneous or brought out by moving the eyeballs and mental symptoms, such as laughing or crying without cause, usually soon develop. Optic atrophy, especially affecting the temporal half of the disk, is a characteristic symptom. Muscular atrophy, tremor of head and legs, diminution or loss of sensation, paresthesia, paralysis of various cranial nerves, apoplectiform or epileptiform attacks, dementia, and incontinence of urine and feces, may all occur.

Atypical cases (the formes frustes of Charcot) are not infrequent and may resemble lateral sclerosis, posterolateral sclerosis, transverse myelitis, or appear as a gradually developing hemiplegia. Careful examination will usually reveal some of the symptoms mentioned above, especially atrophy or pallor of the temporal halves of the optic nerves.

Conditions to Be Differentiated from Multiple Sclerosis

Multiple sclerosis must be distinguished from:

Cerebrospinal syphilis
Hereditary ataxia
Paralysis agitans
Disseminated myelitis
Diffuse sclerosis and pseudosclerosis
Dyssynergia cerebellaris progressiva
Brain tumor
Cerebral palsies of children
Arteriosclerosis
Hysteria.

CEREBROSPINAL SYPHILIS.

In cerebrospinal syphilis, nystagmus, scanning speech, intention tremor, and optic atrophy confined to the temporal halves of the disks would be very unusual. Severe headache is usually a symptom, and in a large proportion of cases there would be a large increase of lymphocytes and a positive Wassermann reaction in the cerebrospinal fluid. A positive luetin reaction may be obtained. Ophthalmoplegia interna is more common in syphilis than in multiple sclerosis. Optic neuritis may be present.

HEREDITARY ATAXIA.

Friedreich's form of hereditary ataxia is distinguished by absence of the knee jerks, and of optic atrophy and the presence of more marked ataxia, talipes, scoliosis, and possible heredity.

The *cerebellar form* is more difficult to differentiate; in it, however, spastic paraplegia is not pronounced and scanning speech is not present. It usually develops about puberty.

Paralysis agitans.

Paralysis agitans develops usually in old age; the tremor is more or less constant and ceases during movement of the limb. True spastic paralysis is not present.

Disseminated Myelitis.

Disseminated myelitis develops acutely, often associated with fever. It follows an infection of some sort. Optic neuritis may be present. Otherwise the symptoms may be very similar.

Diffuse Sclerosis and Pseudosclerosis.

The symptoms of diffuse sclerosis and pseudosclerosis are given below.

Dyssynergia cerebellaris progressiva.

Under this title or that of progressive cerebellar tremor, Hunt has described a series of cases in which there was coarse tremor of the intention type, associated with symptoms of cerebellar disease, i. e., asynergy, adiadochokinesis, etc. (p. 582), and absence of spastic paralysis, optic atrophy, and other symptoms characteristic of multiple sclerosis.

Brain Tumor.

Cases characterized by a gradually developing hemiplegia may be mistaken for brain tumor (p. 720). The diagnosis would depend upon finding either general symptoms characteristic of tumor, Jacksonian convulsions, or of some of the symptoms peculiar to multiple sclerosis.

Cerebral Palsies of Children.

The cerebral palsies of children could only be confused with cases of multiple sclerosis occurring in early life. In the former the symptoms appear either in infancy or develop after an apoplectic onset, which is unusual for multiple sclerosis. Intention tremor, nystagmus, optic atrophy, ophthalmoplegia, and scanning speech, are not usual. The former are common, the latter is rare.

Arteriosclerosis.

Arteriosclerosis may cause scattered areas of softening and symptoms resembling those of multiple sclerosis. Old age, rapid development of the symptoms, and other evidences of arteriosclerosis are in favor of it as a cause.

Hysteria.

Hysteria may coexist with multiple sclerosis. In the former the Babinski reflex, optic atrophy, nystagmus, scanning or slurring speech, are absent.

(*b*) *Diffuse Cerebral Sclerosis*

Diffuse cerebral sclerosis is a rare condition occurring in children and adults. Mental deterioration is rapid, and paresis may be simulated.

Other cases more closely resemble multiple sclerosis. The peculiar ocular symptoms of the latter are usually absent and dementia develops much more rapidly and intensely. Some cases seem to follow traumatism to the head, others—especially in the young—hereditary syphilis.

(*c*) *Pseudosclerosis*

Symptoms somewhat similar to those of multiple sclerosis have occurred in which no lesion was found after death. Such cases have been termed *pseudosclerosis.* The symptoms of others have resembled those of paralysis agitans (p. 758). Recently, the relations of many of such cases and others in which athetoid and choreiform movements have developed to lenticular disease (p. 688) have been shown (Cadwalader, Am. J. Med. Sci., Oct., 1915, p. 556).

Syphilis of the Nervous System

Until the development of the Wassermann reaction and examination of the cerebrospinal fluid, tabes dorsalis and paresis were classed as parasyphilitic diseases. In other words, syphilis acted as an etiological factor in some obscure and indirect way and antisyphilitic medication was of no avail. We now know that these diseases are directly due to syphilis, and antisyphilitic medication if properly administered in the former at least may be of service.

Forms.—Syphilis of the nervous system can be divided into two classes:

(a) Cases in which the lesions are due to a round cell infiltration and exudation involving the arteries and meninges, or meningovascular or exudative syphilis.

(b) Cases in which there is degeneration of nerve fibers and cells, or parenchymatous or degenerative syphilis.

The former comprises what formerly was alone classified as syphilis of the nervous system, and gives rise to various symptom complexes due to either disease of the blood vessels or gummatous exudation, such as apoplexy (usually thrombotic of either brain or cord), meningitis, and gummatous tumor in brain or cord.

The latter comprises paresis, tabes dorsalis, possibly some cases of spinal muscular atrophy and amyotrophic lateral sclerosis and some cases of epilepsy and optic atrophy.

The degenerative form of the cerebral palsies of children may be due to hereditary syphilis.

(*a*) *Exudative Syphilis*

Symptoms.—The symptoms of the exudative type may be due to either hereditary or acquired syphilis, and the latter may appear at any time after the appearance of the initial lesion—even while the secondary symptoms are still present. The usual period is between the third and tenth year after infection.

It has been noticed that those who develop syphilis of the nervous system frequently have had very mild secondary symptoms. It is also important to remember that the cerebrospinal fluid may show a positive Wassermann reaction during the secondary period, and such cases should be treated until it becomes negative. Therefore it is well to have this fluid examined even if symptoms pointing to the nervous system are not present.

As the lesion may be present in any part of the nervous system the symptomatology is varied and any form of system or diffuse disease may be simulated. Dana has well summarized the *common symptoms and their anatomical causes* in the following table:

I. *Prodromal Syphilis.*

II. *Meningovascular Syphilis, Hereditary and Acquired.*

Clinical Symptoms	*Anatomical Change*
(*a*) *of the brain*	
Severe headache, vomiting, vertigo, mental dullness, irritability, attacks of somnolence or coma, convulsions, cranial nerve palsies, optic neuritis, hemiplegia, brain stem and bulbar palsies.	Syphilitic meningitis, arteritis and phlebitis.
(*b*) *of brain and cord*	
Many of the brain symptoms as above, spastic paraplegia, etc.	Meningitis; diffuse, disseminated or localized, meningomyelitis.
(*c*) *of spinal cord*	
Paraplegia, Brown-Séquard paralysis, muscular atrophy, spastic paraplegia and ataxia.	Meningomyelitis, gumma, localized softenings from obliterative arteritis.
(*d*) *of nerve roots and nerves*	
Cranial nerve palsies, cauda-equina symptoms, local palsies of peripheral nerves, muscular atrophy.	Root neuritis, gummatous neuritis.

III. *Parenchymatous Syphilis, Hereditary and Acquired.*

(a) of the brain

Paresis, bulbar palsy.	Meningo-encephalitis.

(b) of the spinal cord

Tabes dorsalis, spastic tabes, muscular atrophy.	Meningomyelitis.

(c) of the brain and cord

Taboparesis.

IV. *Combinations of II and III.*

1. Prodromes

The prodromal signs are very important, as their recognition, if it leads to prompt treatment, may avert more serious conditions.

Symptoms.—The most important of these is *headache* which is often most intense and worse at night. If the symptoms are purely spinal, this of course is absent. Other symptoms are transient cranial nerve palsies, vertigo, epileptic convulsions, paresthesias of the head, somnolence, symptoms of neurasthenia and mental symptoms, either depression, or mania. The latter two are especially apt to precede tabes dorsalis and paresis.

2. Cerebral Syphilis

If the symptoms above mentioned continue, any of a number of others may appear. The patient may be excessively drowsy during the day and wander about the house in an aimless way at night; stupor may deepen into complete coma. Optic neuritis is not uncommon, and as gummatous infiltration is especially apt to occur in the meninges at the base, paralysis of various cranial nerves, *especially the motor nerves of the eyeball* (p. 632), is of frequent occurrence. Lack of response of the pupils to either light or in convergence is very characteristic of syphilis.

Apoplectic attacks, usually due to thrombosis, are of frequent occurrence, and such occurring in a young person in whom the influence of the infectious fevers, or in whom no cause for embolus exists, is usually due to syphilis. A localized gumma causes the symptoms of tumor. If the meninges at the vertex are involved, epileptiform convulsions, which may be of the Jacksonian type if the cortical centers are irritated, are usual. Hypochondria and mental depression may persist even after the objective symptoms have disappeared. Usually the onset of symptoms is gradual but they may develop acutely with coma.

3. Spinal Syphilis

Spinal syphilis may occur as acute myelitis, either transverse or disseminated, spinal muscular atrophy, or spastic paraplegia. A common symptom complex is that known as *Erb's type of syphilitic spinal paralysis.* This usually begins with slowly increasing weakness and stiffness of the legs. A peculiar feature of the stiffness is that it is not apparent when the limbs are moved passively, but appears when the patient attempts to walk. The gait is of the ataxic paraplegic type, the deep reflexes are increased, and the Babinski reflex is present. There is usually difficulty either in passing urine or holding it, and the sexual power is lost. Sensory symptoms, excepting paresthesia of the limbs and back, are not usual. The lesion is a thrombosis occurring in the vessels supplying the lower half of the spinal cord and involving chiefly the posterior columns and posterior part of the lateral columns.

If a localized gumma is the lesion the symptoms are those of spinal tumor or the cauda equina may be affected. Gummatous meningitis involving the posterior roots may cause symptoms simulating tabes dorsalis.

4. Cerebrospinal Syphilis

Cerebrospinal syphilis consists of any combination of the symptoms mentioned under cerebral and spinal syphilis.

5. Syphilis of the Peripheral Nerves

Syphilis of the peripheral nerves usually occurs as an involvement of the nerve roots, either cranial or spinal, in a gummatous meningitis. Multiple neuritis has, however, been described but is rare. When the nerve roots are attacked the symptoms are those of irritation followed by paralysis, either sensory or motor, according to the roots affected.

Diagnosis.—Syphilis of the nervous symptom of the meningovascular type may simulate any of the system or diffuse diseases of the nervous system. There is usually, however, something atypical in the development of the symptoms; thus those of an acute myelitis will be preceded by headache or transient cranial nerve palsies or other of the prodromal symptoms (*supra*), or symptoms of conditions which usually occur late in life, appear early, for instance, as apoplexy. The diagnosis frequently will depend on the history of previous infection and examination of the blood for the Wassermann reaction and of the cerebrospinal fluid for an increase in globulin and the number of lymphocytes.

It must be remembered that examination of the blood alone is not sufficient, as a positive reaction may be present, owing to previous infection, but the nervous disease present is not due to syphilis. If it is positive with the cerebrospinal fluid the evidence is clear. Unfortunately it is not always present when the symptoms are due to syphilis (p. 758), but an increase in the cell count is unless the lesions are purely vascular. An increase in the cell count may be due to meningitis from other causes, but the presence of lymphocytes is in favor of syphilis. It must be remembered that previous medication may render these tests negative. Clinical symptoms and the results of therapeutics may be the sole guides.

This type may also have to be distinguished from the principal forms of the parenchymatous type:

Tabes dorsalis
Paresis.

Tabes dorsalis.

The presence of the Argyll-Robertson pupil, primary optic atrophy and trophic symptoms are in favor of tabes. In this also the luetin test is more apt to be positive, the cell count averages less and the Wassermann reaction is less likely to be positive than in meningovascular syphilis (p. 758).

Paresis.

In paresis headache rarely occurs; delusions of grandeur when present are characteristic; ocular nerve palsies are not common. The cell count is usually low; the Wassermann reaction is positive in a higher percentage of cases in both fluid and blood serum than in either tabes or meningovascular syphilis; the luetin test is positive in a higher percentage of cases, and the colloidal gold test is practically always present, while in tabes it is always absent and is present in a small percentage of cases of meningovascular syphilis (p. 758).

(*b*) *Parenchymatous Syphilis*

1. General Paresis

(*Dementia paralytica, General Paralysis of the Insane*)

Etiology.—This disease is always due to syphilis—either hereditary or acquired—usually the latter. Abuse of alcohol, prolonged mental strain or excitement, the neuropathic diathesis and sexual excesses are predisposing causes. It is comparatively rare in women.

Symptoms.—The pathological condition is a meningo-encephalitis shown by thickening of the membranes and their adherence to the cortex, atrophy of the convolutions, areas of either softening or hemorrhage and either softening or hardening of the brain. There is a round cell infiltration of the arteries; the perivascular spaces are dilated and filled with these cells; the ganglion cells are more or less degenerated as may also be the white fibers. Degeneration of either the lateral or posterior columns of the cord or both is usually present. As has been previously stated tabes dorsalis may coexist (taboparesis).

The symptoms may develop acutely with an apoplectiform or epileptiform seizure. In the great majority of cases the development is gradual and is preceded by a prodromal period during which the patient may be thought to have neurasthenia, as he suffers from insomnia, is irritable, easily fatigued mentally and physically, and may have some failure of memory. In other cases a change in disposition is noted: a formerly temperate man becomes intemperate, he neglects his business, commits sexual excesses and spends money foolishly. During this period there may be spells of mental depression.

Patients presenting such symptoms should be examined for the presence of the characteristic physical symptoms, which are Argyll-Robertson pupils, optic atrophy, either absent, diminished or increased knee jerks, more or less physical weakness, tremor of the hands, of the facial muscles and of the tongue.

The latter is of a peculiar type, the tongue when protruded going in and out of the mouth like the piston of an engine. The speech becomes halting and slurring, syllables being frequently omitted. There is especial difficulty with labials and linguals and in cases where there is not apparent change to ordinary observation they may be brought out by causing the patient to pronounce test sentences or phrases, such as "truly rural," "cavalry brigade," "Peter Piper picked a peck of pickled peppers," and so on. The handwriting is usually tremulous, and syllables and words may be omitted. In some cases the gait may be like that of tabes; in others it may be somewhat spastic or ataxic paraplegic. The lines of the face become smoothed out. Weight may be lost at first, but is often gained later and the appetite may be voracious.

Tabes dorsalis and paresis may be present in the same patient (taboparesis). During the course of the disease apoplectiform or epileptiform seizures may occur. Death may occur during such attacks.

The *mental state* is usually one of exaltation and the so-called "delusions of grandeur" develop. Thus the patient believes he is the strongest man in the world, and that he has untold millions of dollars. Many patients may show only a feeling of well-being and indifference; thus if asked how they feel will always answer "first rate" or "fine." While a family may be dependent upon him for support, the fact that he is not working does not worry him. A small proportion instead of being exalted are depressed and hypochondriacal, and worry about their condition. This may alternate with the exalted state. Outbreaks of great maniacal excitement may occur during the course of the disease. There is always marked failure of memory. Hallucinations are rare and when they occur are often due to some toxic condition, as uremia or alcohol.

The *usual duration of the symptoms* before death occurs is three to five years, and during this period remissions may occur in which the mental condition may be apparently normal and the physical condition improve.

Examination of the blood and cerebrospinal fluid is of great importance. In most cases there is a moderate increase of lymphocytes, the presence of plasma cells, and positive globulin and Wassermann tests in the cerebrospinal fluid. The colloidal gold test is positive in a large proportion of cases, more so than in any other form of syphilis of the nervous system, and the Wassermann reaction is usually present in the blood-serum (See p. 758).

Symptoms of *neurasthenia* should always lead to a careful physical examination for the symptoms of paresis. All of them may not be present but some probably will. A careful serological study should be made in all doubtful cases.

Conditions to Be Differentiated from Paresis

Paresis must be distinguished from:

Alcoholic pseudoparesis
Lead encephalopathy
Cerebral syphilis of meningovascular type
Tabes dorsalis
Mania
Multiple sclerosis
Diffuse sclerosis
Chronic bulbar palsy
Tumor of frontal lobes
Terminal dementia.

ALCOHOLIC PSEUDOPARESIS.

Chronic alcoholism may simulate paresis, both physically and mentally. In the former Argyll-Robertson pupils will not be present. The "delusions of grandeur" are not apt to be so pronounced; spastic paralysis does not occur, although ataxia and absent knee jerks may; the cerebrospinal fluid will not give positive results to the tests mentioned above; and recovery may take place.

LEAD ENCEPHALOPATHY.

Chronic lead poisoning may cause tremor and mental symptoms. The blue line on the gums or other evidence of lead poisoning, and often a history of exposure will be found. Examination of the blood and cerebrospinal fluid, as above, is important.

CEREBRAL SYPHILIS OF THE MENINGOVASCULAR TYPE.

Cerebral syphilis of the meningovascular type rarely shows the Argyll-Robertson pupil. Headache is usual. The exalted state is not pronounced, stupor being the more usual condition. The lymphocytes in the cerebrospinal fluid are more greatly increased; the colloidal gold test is less likely to be positive (See p. 758); the luetin test is less apt to be positive. Ocular nerve palsies are more likely to be present and the symptoms are apt to develop in less than ten years after infection; paresis is more apt to develop after ten years. Optic neuritis is not usual in paresis.

TABES DORSALIS.

In tabes dorsalis if mental symptoms do occur, it is not until late in the disease. The peculiar tremors and speech disturbance are not present. Pain is a marked symptom. It must be remembered that tabes dorsalis and paresis are frequently associated.

MANIA.

The mental symptoms of mania, especially *hypomania,* may resemble those of paresis. The physical symptoms are absent.

MULTIPLE SCLEROSIS.

Multiple sclerosis may be mistaken on account of the disturbance of speech; as a rule, this does not resemble that of paresis (*vide*). The mental state is different; nystagmus is present; the tremor is of the intention type.

DIFFUSE SCLEROSIS.

Diffuse sclerosis may simulate paresis very closely. A serological examination will, if it gives the usual results found in paresis, be in favor of that disease.

CHRONIC BULBAR PALSY.

Chronic bulbar palsy may be mistaken, as the speech and tremor of the lips and tongue may resemble paresis. There will be, however, atrophy of the tongue, no dementia and no delusions. Paralysis of the vocal cords, throat muscles and tongue will be present.

TUMOR OF THE FRONTAL LOBE.

Tumor of the frontal lobe may cause mental symptoms resembling those of paresis. Sooner or later hemiplegia will usually develop from pressure on the motor centers. If on the left side motor aphasia is likely to be present and perseveration (p. 614) may be found. Serological examination will not give the results peculiar to paresis.

TERMINAL DEMENTIA.

Terminal dementia following apoplexy can hardly be mistaken if attention is paid to the history of the case. In doubtful cases a serological examination will be sufficient.

2. Tabes dorsalis

(*Locomotor ataxia, Posterior Sclerosis*)

Tabes dorsalis is a chronic and progressive disease beginning as a mild meningitis, affecting the posterior roots as they pass through the meninges after leaving the ganglia, and followed by degeneration first of the tract of Lissauer, then the columns of Burdach and Goll, and finally other afferent columns, as Clarke's and Gower's. Various cranial nerves also may suffer, especially the optic. While the disease principally affects sensory roots and nerves, the anterior spinal roots may rarely suffer and motor nerves of the eyeball rather frequently (p. 623). It usually affects first the lumbosacral roots, but may begin in either the sacral, cervical, or bulbar regions.

Etiology.—It may occur at any age and the cause is always syphilis, either hereditary or acquired, whether admitted or not. Prolonged physical exhaustion, infectious diseases, excessive use of tobacco and trauma may excite the condition in those predisposed by previous syphilitic infection.

Symptoms.—The symptoms are divided into three stages: preataxic, ataxic, and paralytic.

PREATAXIC STAGE.—Those of the first stage are of great importance, as recognition of the disease then and the use of proper therapeutical measures may make a great difference in the future welfare of the patient.

The first symptom in the vast majority of cases is *pain,* usually of the root type (p. 567). The paroxysms are often intense and described as shooting, stabbing, or lancinating; they may be but momentary and may leave a feeling of soreness for a time. In some cases the pain may be more or less constant and confined to one spot. In the ordinary type of case they are first felt in the legs. If the patient is examined at this time lost knee jerks and probably Achilles jerks, the presence of Argyll-Robertson pupils (p. 592) and possibly slight ataxia, as shown by difficulty in walking in the dark or with the eyes closed, standing on one leg or walking backwards, will be found. It is of the greatest importance to remember that the *symptoms above mentioned, especially pain, may precede the development of appreciable difficulty in walking a long time.* In fact, in some cases, ataxia is never present except when shown by delicate tests, as above mentioned.

Other symptoms, which more rarely are first complained of, are: diplopia and paralysis of ocular muscles; visceral crises (*infra*); loss of sexual power; difficulty in urinating; becoming easily fatigued (neurasthenic); swelling of the joints (arthropathies, *infra*); and feelings of numbness and as if walking on something soft.

Confirmatory symptoms may be analgesia of the ulnar nerve when pressed upon (Biernacki's sign) and of the testicle.

In *bulbar tabes* the pain would be in the course of the fifth nerve and may be mistaken for trigeminal neuralgia.

In *cervical tabes* it would be in the arms, and the knee jerks may be preserved and the biceps jerks lost.

In *sacral tabes* bladder crises (*infra*) may be an early symptom, the knee jerks being preserved and the Achilles jerk lost.

At this time also the cerebrospinal fluid will probably show a considerable increase in cells of the lymphocyte type (30 or more), and a positive globulin test. The Wassermann reaction may be positive in the cerebrospinal fluid while it is negative in the blood serum. This, however, may be reversed or it may be positive in both. In cases where the Wassermann test is negative the luetin test of Noguchi may be positive. It is so in a larger proportion of cases than is the Wassermann (See pp. 755, 758).

Ataxic Stage.—In the ataxic stage the symptoms above mentioned persist and often increase in severity; the Romberg sign (p. 587) is marked and the gait becomes characteristic (p. 586). More or less muscular hypotonia causing relaxation of the joints will be found, so that in extreme cases the patient can do the tricks of the contortionist (Fig. 169). The pupils are more or less contracted (myotic) and unequal, and examination of the optic nerve may show more or less primary atrophy. This sometimes occurs early and advances rapidly to blindness. Such cases are known as *ocular tabes,* and other symptoms, excepting pain, are usually not marked.

At this stage there are apt to be areas of analgesia, especially on the legs and about the body at the nipple line; they are usually of the segmental type. A characteristic sensory symptom is known as "delayed sensation"; in this there is an appreciable interval, often several seconds, between the stimulus and its perception.

Other symptoms of importance but which do not occur so frequently are: *visceral crises,* by which are meant paroxysms of pain referred to different viscera. The most common is the gastric, in which the pain is referred to the stomach; there is incessant vomiting and hyperchlorhydria. Others occurring with more or less frequency are *laryngeal crises* in which there are dyspnea, continuous coughing, and noisy inspiration, *bladder crises* characterized by pain in that region and very frequent urination. The attacks occur at intervals ranging from several days to months and usually last three or four days, and *ocular crises* in which there are severe pain in the eyes, lacrimation, photophobia, blepharospasm, and at times visual hallucinations. Such symptoms resemble one form of migraine (p. 772).

Nephritic, rectal, urethral and *clitoral crises* also occur.

Trophic disturbances, as arthropathies (Fig. 170), *perforating ulcers,* usually situated back of the big toe (Fig. 171), *brittleness of the bones,* so that they break almost without cause, *thickening of the nails, onychia, and eruption of herpes,* occur more or less frequently.

Degeneration or Inflammation of Peripheral Nerves.—Among the cranial nerves the auditory is sometimes thus affected, causing deafness, vertigo and tinnitus. When the anterior spinal roots are affected muscular atrophy, often of the Aran-Duchenne type (p. 694), occurs.

Loss of Muscle sense is shown by inability to recognize the position of the limbs or the direction of passive movements (p. 580).

Paralytic Stage.—In the paralytic stage loss of muscular power appears, and the patient becomes bedridden. Previously the disability has been due to inability to control the limbs and not to loss of strength.

Retention of urine, with often the development of cystitis, may be symptoms in this stage.

Mental symptoms may be present. In this connection it must be remembered that paresis and tabes may occur together (p. 749).

Diagnosis.—Examination of the cerebrospinal fluid now may be negative, as the active process is apt to have ceased and only its effects evidenced by degeneration of nerve cells and fibers remain.

Conditions to Be Differentiated from Tabes dorsalis

Tabes dorsalis must be distinguished from:

Spinal syphilis of meningovascular type
Paresis
Multiple neuritis due to alcohol, diabetes, and tobacco
Sciatica
Ataxic paraplegia
Combined sclerosis
Friedreich's ataxia
Cerebellar disease
Visceral disease affecting appendix, gall-bladder, etc.

SPINAL SYPHILIS OF MENINGOVASCULAR TYPE.

Syphilitic meningitis may involve the posterior nerve roots and cause symptoms which simulate tabes. A very high cell count (over 100) and rapid amelioration of the symptoms by treatment are in favor of a meningitis. In such cases also the luetin test is less apt to be positive than is the Wassermann (See p. 758).

PARESIS.

Tabetic symptoms are frequently found associated with paresis. The peculiar speech, tremor of hands and tongue, occurrence of delusions and dementia found in paresis, are not present in pure tabes.

Multiple Neuritis Due to Alcohol, Diabetes and Tobacco.

In multiple neuritis there will be early development of paralysis and muscular atrophy and, with the exception of the lead and diphtheritic forms, pain and tenderness over the affected nerves. There will be absence of pupillary symptoms (Argyll-Robertson).

Alcoholic and diabetic neuritis are especially apt to simulate tabes, but attention to the history, examination of the urine and the presence of symptoms of neuritis should make the diagnosis plain.

Excessive use of tobacco may also cause symptoms simulating tabes. I have seen several cases in which there was lost knee jerk, pain in the legs and a gait resembling that of tabes. Such cases are rare; they will have the eye symptoms caused by tobacco (p. 599). The spinal fluid will be normal.

Sciatica.

Tabetic pains have often been diagnosed as sciatica (p. 665). In this there is tenderness over the nerve; the knee jerk is preserved, the pain is usually unilateral; Laseque's sign is present (p. 665); the peculiar ocular symptoms and evidences of ataxia are absent.

Ataxic Paraplegia.

Ataxic paraplegia, or posterolateral sclerosis, owing to the ataxic gait, may be mistaken. In it, however, the knee jerks are increased, the Babinski is present, some spasticity is present, eye symptoms are absent.

Combined Sclerosis.

Owing to the intense paresthesia and pain, subacute combined sclerosis may be mistaken. In it, however, there are no eye symptoms and in the earlier stages the legs are spastic, the knee jerks increased and the Babinski reflex present.

Friedreich's Ataxia.

Friedreich's ataxia, a form of posterolateral sclerosis, occurs in early life; it may be hereditary; there are disturbances of speech, nystagmus and the Babinski reflex.

Cerebellar Disease.

Cerebellar disease has a different form of gait (p. 582); pain in the limbs is absent; knee jerks may be absent or increased; Argyll-Robertson pupil is not present. If a tumor, papilledema may be present, and if the lateral lobes are affected, dysmetria and a diadochokinesis are present (p. 583).

Visceral Disease Affecting Appendix, Gall-bladder, etc.

Visceral crises have often been mistaken for visceral disease. Careful examination should be made when paroxysmal attacks of pain anywhere occur from symptoms of possible tabes.

	Tabes dorsalis	Multiple Neuritis	Sciatica	Posterolateral Sclerosis	Cerebellar Disease
Pain	Shooting and momentary, no tenderness over nerves	Constant, increased by pressure over affected nerves	Constant, increased by motion, tender over nerve and at sciatic notch	Dull pain in region of sacrum at times. Paresthesia sometimes	No pain in limbs. If tumor, may be headache or pain in trigeminal nerve
Gait	Feet wide apart, eyes looking at ground. Joints related	Weakness, steppage gait (p. 672)	Favors affected limb, leans body away from it	Mixture of ataxia and spasticity	Like drunken man, falls to one side or other, sometimes forward or backward
Eyes	Unequal myotic pupils, primary optic atrophy. Sometimes diplopia, Argyll-Robertson pupil	No changes as a rule. Optic neuritis and paralysis of extraocular muscles may occur	No changes	No changes, except in Friedreich's ataxia, when there is nystagmus	Nystagmus. If tumor possibly papilledema. Paralysis of ocular muscles may be present
Reflexes	Tendon jerks, especially knee and Achilles, usually lost. No Babinski	Tendon jerks in affected limbs lost. No Babinski	Achilles jerk often lost, no other changes	Knee jerk present or increased Ankle clonus sometimes. Babinski present. In Friedreich's ataxia knee jerk is lost, ankle clonus never present	Knee jerks may be absent, present or increased
Trophic Changes	Changes in joints, bones, sometimes. Muscular atrophy rare	Muscular atrophy common. Glossy, shining skin	No changes except in cases of long standing, then possibly atrophy	No change as a rule. Muscular atrophy at times in Friedreich's ataxia	None
Mental Symptoms	Rare except in later stages or when associated with paresis	Frequent in alcoholic form, sometimes in that due to lead	None	None	None, except in later stages of tumor there may be stupor or when lack of cerebellar development occurs in imbeciles
Examination Cerebrospinal Fluid	Increase in number of cells. Globulin reaction, sometimes Wassermann reaction. In old cases it may be apparently normal. Luetin test positive	Normal	Normal	Normal, unless due to syphilis, when some increase in cell count and Wassermann may be present	Normal, unless lesion is syphilitic

The following table, prepared by Kaplan for Dana's "Textbook of Nervous Diseases," eighth edition, shows well the usual results of serological tests in the various forms of syphilis of the nervous system.

SEROLOGICAL FORMULAE FOUND IN SYPHILITIC NERVOUS DISEASES

	Cells per c.c.	W. R. in C–S. F.	Globulin	Fehling Reduction	Colloidal Gold	W. R. in Blood Serum
I Meningovascular Lues Cerebral, Spinal or Cerebrospinal	Average 80–20 Extremes 0-2000	+in 65 per cent	Excess in 65 per cent	Present in 95 per cent	Present less than 2 per cent	+in 80 per cent
II Tabes dorsalis	Average 35–60 Extremes 0-350	+in 40 per cent	Excess in 33 per cent	Present in 99 per cent	Absent	+in 65 per cent
III Paresis	Average 18–35 Extremes 0-250	+in 85 per cent	Excess in 75 per cent	Present in 100 per cent	Present in 95 per cent	+in 95 per cent
Taboparesis	20–40	+in 85 per cent	Excess in 70 per cent	Present in 100 per cent	Present in 75 per cent	+in 95 per cent

J. General and Functional Diseases

1. Paralysis agitans

Occurrence.—Paralysis agitans, also known as *Parkinson's disease* and *shaking palsy*, usually develops between the ages of forty and sixty-five; it may rarely do so earlier.

Causes.—Fever, prolonged anxiety, overwork, grief, and trauma, may be exciting causes.

Symptoms.—The symptoms may appear on one side first, and usually tremor of the hand is first noticed. This is usually a slow, coarse movement, which ceases momentarily when the limb is moved. Frequently the patient appears to be rolling a small object between the thumb and index finger, and for this reason it has been termed the "pill-rolling tremor." Sooner or later the other side becomes affected. Less frequently tremor appears in the legs, and rarely in the head.

Early in the course of the disease the muscles become rigid, and overaction of the flexor muscles causes the characteristic position shown in Figure 184, i. e., the head and body are bent forward, the fingers flexed on the metacarpal bones, the forearms on the arms, and the knees slightly bent. Muscular movements are slow, but the deep reflexes are not increased as a rule, and the Babinski reflex is not present.

The facies is peculiar, often termed masklike, the lines of expression being smoothed out and the eyelids widely opened and rarely winking (Fig. 184).

In walking the steps are short and shuffling, and often there is a tendency for the body to pitch forward. To prevent this the patient runs in an effort, apparently, to make his feet catch up with the head and shoulders. This has been termed *propulsion*, or *festination* (Fig. 184a). Dull pains in the limbs and feelings of heat are often complained of. Vasomotor disturbances shown by sweating and flushing and elevated surface temperature may be present.

The speech becomes slow and monotonous, and while dementia does not occur, the patient becomes apathetic and often appears to think slowly.

Cases occur in which tremor is absent; then the diagnosis depends on the rigidity, peculiar attitude, gait, and facial expression.

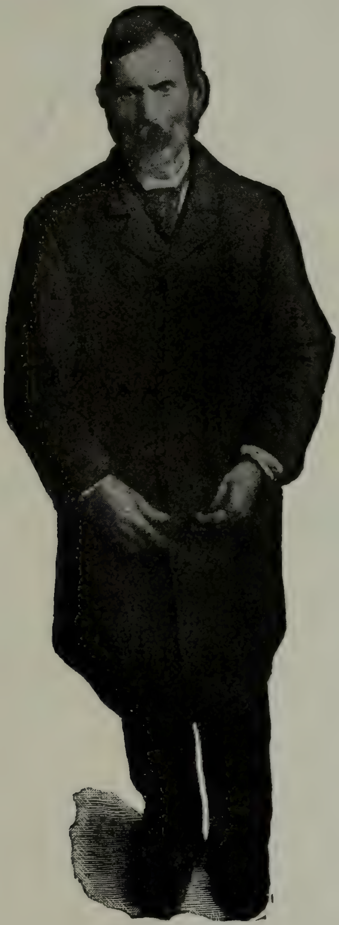

Fig. 184.—Photograph of a Case of Paralysis agitans, Showing the Attitude, the Position of the Hands, and the Facies. (After Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

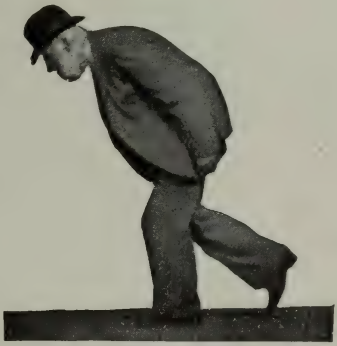

Fig. 184a.—Gait in Paralysis agitans, Showing Propulsion. (After Church and Peterson's "Nervous and Mental Diseases," published by W. B. Saunders Co., Philadelphia.)

Conditions to Be Differentiated from Paralysis agitans

Paralysis agitans must be distinguished from:

Senile tremor
Multiple sclerosis
Postapoplectic tremor
Chorea
Hysteria (See also p. 560).

SENILE TREMOR.

Senile tremor occurs usually in the very old. Tremor frequently affects the head and does not cease—in fact, increases during movement of the limbs. The peculiar rigidity and gait are absent. Old persons with arteriosclerosis may walk with short, shuffling steps, but festination does not occur. The expressionless face may also occur in those with arteriosclerosis.

MULTIPLE SCLEROSIS.

Multiple sclerosis occurs usually in young adults. The tremor increases during exertion; the tendon reflexes are greatly increased; the Babinski reflex is often present; nystagmus and optic atrophy are usually present.

POSTAPOPLECTIC TREMOR.

Postapoplectic tremor consists of tremor affecting the paralyzed arm after an apoplectic attack. It is usually increased during efforts at movement. The reflexes are increased and the Babinski reflex present on the paralyzed side. The gait is that characteristic of hemiplegia (p. 708). There will be a history of an apoplectic attack (p. 621).

CHOREA.

Chorea sometimes occurs in old people. The choreiform movement is an irregular, non-purposive movement, affecting first one group of muscles, then another. Muscular rigidity, the peculiar gait and attitude are wanting.

HYSTERIA.

Hysteria may simulate any disease. Hysterical tremor is usually increased during movement.

There is usually a history of sudden onset following a shock or emotional disturbance. Sensory paralyses and hysterogenic zones are usually present (p. 778). It may frequently be made to cease by suggestion or mental impression. The peculiar attitude and gait of paralysis agitans do not occur in hysteria.

2. Choreiform Affections

These are also termed myoclonias. Myoclonia is a term used to designate clonic spasms or twitching of muscles as distinguished from *myotonia*, which means tonic spasm of a muscle or muscles. *Myoclonus* means a twitching muscle. The myoclonias comprise Sydenham's chorea, hereditary chorea, spasmodic tics, and those characterized by fibrillary twitchings, as paramyoclonus multiplex.

(*a*) *Chorea of Sydenham*

The chorea of Sydenham, also known as *St. Vitus' dance, St. Anthony's dance, infectious myoclonia, infectious chorea,* and *chorea minor,* is the most common of these.

Occurrence.—It most commonly occurs in children, between the ages of five and fifteen years, affecting girls more frequently than boys; it may occur in adults and old people.

When chorea occurs in adults, males are more commonly attacked than females. It is apt to occur in those who have had the disease in childhood. It is due to the same causes as in children, but is more apt to become chronic. When it occurs in old people, it is known as *senile chorea.* These forms must not be confused with hereditary chorea (*vide*).

Etiology.—It is of infectious origin, the exact nature of which is not positively known (the Micrococcus viridans in some cases). One attack seems to predispose to others. Overwork and nervous strain at school, anemia, and malnutrition are also predisposing factors.

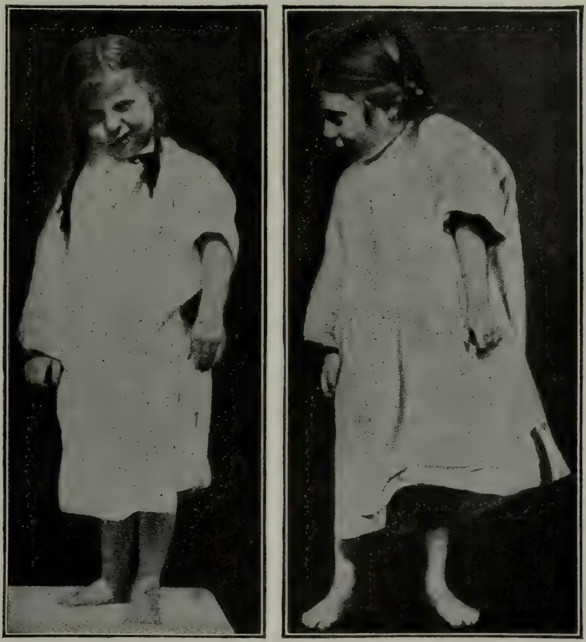

Fig. 185.—Chorea, Showing Grimace and Shoulder Movement. (After Jacobsohn.)

The exciting causes may be either fright, prolonged worry, physical injury, pregnancy, acute infectious fevers, septic infection, acute articular rheumatism, acute tonsillitis, and chronic disease of the tonsils.

Symptoms.—The symptoms may develop suddenly, but usually do so gradually. Twitching of the fingers on one side and of muscles of the face is usually noticed first. This soon involves both sides and other muscles until there are continual, irregular movements of the limbs, facial muscles, sometimes of the tongue, laryngeal muscles, and those of respiration (Fig. 185).

The speech becomes indistinct; grunting sounds may be made; breathing is irregular; things are dropped from the hand; and the patient becomes irritable and hysterical. The movements usually cease during sleep, but in severe cases may continue and prevent proper rest.

The attack usually reaches its maximum in about two weeks, but it may continue from three weeks to three months or longer. In some cases the symptoms may be milder than those just described and speech may not be affected; in others, however, they may be much worse, and in the so-called *chorea insaniens* there is marked fever, violent movements, and delirium. This form is especially apt to occur during pregnancy. In some cases one side may become actually weak. Such are known as *paralytic chorea.*

Other symptoms which may occur are dull aching of the limbs, skin eruptions, as purpura, herpes, and erythema nodosum, subcutaneous fibrous nodules, nocturnal enuresis, absent or diminished knee jerks, or the so-called *"tonic reflex,"* i. e., when the patellar tendon is struck and the foot flies up, it remains there for a few seconds instead of dropping back at once.

Associated Conditions.—Endocarditis occurs in a relatively large number of cases, and chorea is a common cause of heart murmur. In 110 cases examined by Osler two years and more after an attack, 64 showed some evidence of organic heart disease.

All murmurs heard during the course of the disease are not necessarily due to organic disease. Hemic murmurs are frequently due to the anemia which frequently coexists. Pericarditis occasionally occurs.

The diagnosis should not be difficult.

Conditions to Be Differentiated from Chorea of Sydenham

It must be distinguished from:

Hysterical chorea
Athetosis and postapoplectic chorea
Spasmodic tic
Paramyoclonus multiplex
Hereditary chorea.

HYSTERICAL CHOREA.

In hysteria movements simulating those of chorea may occur. In such cases these are much more sharp and quick, often resembling the contraction of a muscle stimulated by electricity. Other stigmata of hysteria will be present. It must be remembered that hysteria and chorea may coexist.

Athetosis and Postapoplectic Chorea.

Athetoid movements and the choreiform movements which sometimes occur in the paralyzed limbs after an apoplectic seizure may be mistaken, especially if paralysis is not marked. Some evidence, such as increased knee jerks, Babinski reflex, and other evidence of central neuron lesion will be present.

Paralytic chorea can be distinguished by the fact that these symptoms are absent and the limbs flaccid. Athetoid movements are more rhythmic than those of chorea and are confined always to the same muscles (p. 621).

Spasmodic Tic.

The distinguishing features of spasmodic tic are given on page 765.

Paramyoclonus multiplex.

In paramyoclonus multiplex the muscular contractions are quick, and produce little or no motion of the limb. They are confined usually to muscles of the trunk and limbs, those of the face not being affected. There are no disturbances of speech (p. 766).

Hereditary Chorea.

Hereditary chorea may be mistaken in cases in which Sydenham's chorea occurs late in life. It may be difficult at first to distinguish the two without a history of a similar condition in other members of the family. In hereditary chorea, dementia, usually progressive, soon develops, and there are peculiar disturbances of gait and speech (*vide.*)

(*b*) *Hereditary Chorea*

Hereditary chorea, also known as *Huntington's chorea, chronic progressive chorea,* and *degenerative myoclonia*, occurs in successive generations of a family.

Symptoms.—The symptoms usually appear between the ages of thirty and forty.

Before the appearance of the movements, the patient may complain of nervousness, being easily fatigued, and loss of ambition. Difficulty may be noticed in performing fine movements, such as writing.

The choreiform movements first appear in the upper limbs, and resemble those of Sydenham's chorea. In time they become more or less general. When fully developed they are more rhythmic and extreme than those of the latter.

Whole groups of muscles contract, and this causes a peculiar gait. The patient will take a few steps normally, then a long step, the other leg being brought up to it quickly, followed by one or two hops. This causes a peculiar, undulating motion. The speech is early affected and becomes very indistinct; swallowing may also be difficult. Mental depression, sometimes delusions and dementia, develop sooner or later.

Conditions to Be Differentiated from Hereditary Chorea

This disease has been mistaken for:

Dystonia musculorum deformans
Sydenham's chorea.

DYSTONIA MUSCULORUM DEFORMANS.

This condition occurs in Jews, is not hereditary, and consists of tonic and clonic spasms of the muscles about the pelvis (*vide*).

SYDENHAM'S CHOREA.

Sydenham's chorea occurring in adults is mentioned on page 761.

(c) *Spasmodic Tic*

Spasmodic tic, also known as *habit chorea, habit spasm, motor tic,* and *palmus,* has been defined by Meige and Feindel[1] to be "a coördinated purposive act provoked in the first instance by some external cause or by an idea; repetition leads to its becoming habitual, and finally, to its involuntary production without cause and for no purpose, at the same time as its form, intensity, and frequency are exaggerated. It thus assumes the characters of a convulsive movement, inopportune and excessive; its execution is often preceded by an irresistible impulse, its suppression associated with discomfort. The effect of distraction or volitional effort is to diminish its activity. In sleep it disappears. It occurs in predisposed individuals, who usually show other evidences of mental instability."

The definition well describes the condition. It occurs in those with a neuropathic ancestry and those who themselves are subject to hysteria, neurasthenia, and other psychoneuroses. It is often excited by an effort to relieve some peripheral irritation; thus a tic of the tongue, in which the patient continually at intervals protruded the tongue, was caused by moistening with the end of the tongue a sore on the chin. The patient had had an attack of neurasthenia previously.

Occurrence.—The condition may develop at any age and any group of muscles may be affected. In time other muscles and movements than those originally involved may develop the condition.

Varieties.—There have been described facial or mimic tics, tics of the nose or sniffling tics, tics of the lips or sulking tics, of the tongue or licking tics, of the neck or nodding tics, of the trunk muscles, of the arms, of the hands or scratching tics, of the legs or leaping tics, bowing, whistling, coughing and sobbing tics.

Any voluntary and purposive act may be imitated. The movements are usually quicker than those of the corresponding normal act.

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[1] "Tics and Their Treatment."

(*d*) *Tic convulsif*

Tic convulsif, or *Gilles de la Tourette's disease,* is a modification of simple tic, and consists of similar movements plus mental symptoms.

Symptoms.—These are: *coprolalia,* or the involuntary exclamation of obscene and profane words; *echolalia,* in which names or sentences heard are repeated; *echopraxia,* in which movements made by other persons are imitated.

Fixed ideas, morbid impulses, and various hysterical phenomena may also be present.

Conditions to Be Differentiated from Tic

Tic must be distinguished from:

Sydenham's chorea
True spasm
Paramyoclonus multiplex.

Sydenham's Chorea.

In Sydenham's chorea the movements are irregular, non-purposive, and not confined to any one group of muscles. First one group of muscles will contract, then another, and so on. Speech is often affected. The mental attitude of the patient is different from that of his normal state.

True Spasm.

True spasm has been defined by Meige and Feindel to be motor reaction, consequent on stimulation of some point in a reflex spinal or bulbospinal arc (p. 587). It is therefore due to actual irritation. The points of difference have been well summed up by Patrick:

Tic	Spasm
Uncommon.	Not so common as tic.
Occurs in nervous or neuropathic individuals.	Disposition or temperament has nothing to do with spasm.
Is under the control of the will to some extent and always subject to involuntary control by strong, emotional, or intellectual preoccupation.	Is not under control of the will either voluntarily or involuntarily.
Is a physiological disorder.	Is an anatomical disorder.
The patient can always repeat or imitate his tic movement and another person can usually do it.	Voluntary stimulation is impossible.
It always involves all the muscles taking part in any one physiological movement.	In the beginning may be confined to part of a muscle or of the muscles taking part in a movement (fascicular contractions).

PARAMYOCLONUS MULTIPLEX.

The characteristics of this disorder are given on pages 531-532 and *infra.*

A form of myoclonus is associated with epilepsy—the so-called *myoclonus epilepsy* (p. 769).

Peculiar forms of tic in which there are jumping movements, are endemic in various localities and probably have a hysterical basis. They occur in Maine and Canada, and are there known as *"jumpers"*; in Russia they are known as *"myriachit,"* and in Java and the Malay peninsula they are known as *"latah."*

(*e*) *Paramyoclonus multiplex*

Paramyoclonus multiplex, or *myoclonus multiplex of Friedreich, convulsive tremor, myospasm, fibrillary myoclonia,* and *myokymia,* is a rare condition characterized by quick, clonic contraction of the muscles of the trunk and extremities. These contractions, while marked, produce little or no motion of the limb. The condition is described on page 531.

As has been said (*supra*), similar contractions occur in some epileptics. There is also a family type—that of Unverricht.

Other cases seem to be due to a toxemia, and others may have a hysterical basis.

Muscular spasms of a similar nature may be caused by *exposure to intense heat;* but in this case they are usually painful.

Choreiform movements cause motion in the parts affected, as do also the contractions of tic or habit spasm.

In *Jacksonian epilepsy* motion is caused and signs of disease of the motor cortical centers will usually be found.

(*f*) *Dysbasia lordotica progressiva*

Dysbasia lordotica progressiva, or *dystonia musculorum deformans,* or *tortipelvis,* occurs usually in children and young adults of the Jewish race. It consists of tonic and clonic spasms of the muscles of the pelvic girdle, associated or not with twitchings of other muscles.

The body is bent forward, sideways, and twisted. When recumbent, the symptoms are usually not present. Standing, and especially walking, bring on the spasms. Marked lordosis in the lower dorsal and upper lumbar region is caused with marked protrusion of the buttocks. The gait is peculiar, and has been termed "the dromedary gait." There is no mental deterioration.

3. Epilepsy

Epilepsy is an affection of the brain characterized by paroxysmal attacks of unconsciousness, with or without motor convulsion, and sometimes accompanied with peculiar mental disturbances.

Loss of consciousness with general motor convulsion, is known as *"grand mal"*; brief loss of consciousness without convulsion, as *"petit mal"*; attacks in which the patient runs a short distance, as *"procursive epilepsy"*; and those in which there may be mental excitement and acts of violence committed or in which he may for a number of days perform accustomed and natural acts, are known as *"psychical epilepsy"*; *"epileptic automatism,"* or the *"epileptic equivalent."*

JACKSONIAN EPILEPSY.—Jacksonian epilepsy is a special form, usually due to some irritative lesion of the motor cortex (p. 614), i. e., tumor, syphilitic meningitis, depressed fracture, cyst following cerebral apoplexy, or sclerosis following encephalitis.

It is characterized by clonic spasms, occurring at more or less frequent intervals, affecting a group of muscles or a limb.

The muscles in which the spasm first appears have a direct relation to the location of the lesion. The spasms may spread to other muscles, and in some cases after a decided interval, become general; this is not unusual. Consciousness is seldom lost; if it is, it is only in those cases in which general convulsions develop.

EPILEPTIFORM SEIZURES.—Epileptiform seizures may be divided into two classes, viz.: those for which no cause can be found—the so-called *idiopathic epilepsy*—and those due to some ascertainable origin or toxic disturbance.

The majority of cases in the former class develop in early life. Of 1,450 cases analyzed by Gowers, 442 had their first seizures before the tenth year and 75 per cent before the twentieth year. In this connection it must be borne in mind that a fair proportion of those developing the disease in childhood have had a cerebral lesion, either hemorrhagic or inflammatory, and that these cases belong to the latter class. Such cases are usually hemiplegic or diplegic (p. 689).

Predisposing Causes.—In the former class either alcoholism, syphilis, or a neurotic diathesis in the parents, are predisposing causes. Rachitic children are also predisposed.

EXCITING CAUSES.

Trauma, exclusive of that causing visible brain injury, mental shock or fright, acute infectious fevers, reflex irritations, as worms, teething, adherent prepuce, and adenoids, may act as exciting causes in those predisposed.

With the exception above noted, cases of the latter class are more apt to occur in adult life. It may be put down as a clinical rule that epileptiform convulsions developing in a person past thirty-five years of age are rarely idiopathic.

In this connection it must be borne in mind that convulsions may have occurred in childhood and then ceased, to reappear after a long interval.

The writer knows of a case in which the time which elapsed was over thirty years. If this can be ruled out the following causes must be considered.

Organic brain diseases, as either tumor,[1] abscess, syphilitic meningitis, traumatism causing fracture of the skull or the result of a previous apoplectic attack.

Toxemias, as alcoholism, lead, uremia, products of intestinal putrefaction (various abnormalities, as kinks, ptosis, patent ileocecal valve, have been found by x-ray examination), syphilis (during the secondary stage convulsions may occur without apparent organic disease of the nervous system), and rarely hyperthyroidism.

Circulatory disturbances, as arteriosclerosis, degeneration of the heart muscle, Raynaud's disease, and as one of the symptoms of Stokes-Adams disease.

Pituitary Insufficiency

Reflex Irritations.—These act rarely in causing the disease, but they may in predisposed children. Their chief importance is in aggravating and increasing the number of convulsions in those who already suffer from the disease. The most important are eye strain, nasal and pharyngeal diseases, as adenoids, adherent prepuce, intestinal worms.

Epileptiform convulsions may also occur during the course of multiple sclerosis, paresis, and dementia precox.

Symptoms.—(1) *Grand Mal.* In a small proportion of cases there may be precursory symptoms present for a day or two previous to the seizure, as either irritability of temper, delusions, hallucinations, vertigo, headache, or voracious appetite.

In a larger proportion (about 50 per cent) a peculiar sensation immediately precedes the convulsion. This is known as the *aura.* It may be a sensation of either tingling, numbness, or a breeze, which begins in the hand or leg and passes upward, vague sensations or pains in the epigastrium, pain in the cardiac region, or palpitation of the heart, giddiness, feelings as if something were wrong.

Special sense aurae have a peculiar significance, as they may be focal symptoms. Thus in lesions involving the uncinate gyrus and vicinity, there may at intervals be the sensation of an unpleasant smell. There may also be sensations of taste associated with chewing movements and a dreamy and confused state which Hughlings Jackson has termed, *"uncinate fits."* Either anosmia or hyperosmia may also be present (p. 632).

Visual aurae, as flashes of light, may be present in lesions of the occipital lobe. If of organic nature there would also be permanent homonymous hemianopsia (p. 600) which would distinguish it from the visual hallucinations, sometimes accompanied by transient hemianopsia which may occur in migraine (p. 772).

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[1] Tumor anywhere in the brain may cause general convulsions, and these have no localizing value, as do those of the Jacksonian type (See pp. 718, 767).

Whether an aura precedes the convulsion or not, there is either a loud cry or peculiar groan; the patient falls, and at first there is a brief period of tonic spasm in which the head is drawn around, the hands clenched, the thumb often being inside the fingers, and the arms flexed. The face is momentarily pale, then becomes livid, and the pupils are dilated and insensitive to light.

This is followed by clonic spasms of all the muscles. Saliva comes from the mouth, which, if the tongue is bitten, is bloody; urine and feces may be discharged.

The convulsion lasts a minute or two, and is followed by a period in which the patient is comatose, the face congested, and the breathing stertorous. He may sleep for some hours, awakening often with a headache or mental confusion.

Transient paralysis may follow an attack. In some patients seizures may only occur at night (nocturnal epilepsy). Occasional wetting of the bed, awaking in the morning with headache and mental confusion are suggestive of this condition. The so-called "night terrors" of children, or "pavor nocturnus," may be due to it.

A number of seizures following each other in rapid succession is known as the *status epilepticus.* During this period fever may be present, and afterward the patient may be weak, confused, and delirious for a number of days. After an attack the patient may be very irritable, have delusions and hallucinations, and sometimes become maniacal.

(2) *Petit Mal.* These attacks may consist of one of the aurae, mentioned above, followed by a brief period of unconsciousness. The patient does not fall but may stop momentarily what he is doing, be observed to become pale and have a staring expression, and the pupils become dilated. After the attack he goes on as if nothing had happened.

In others there may be no aura and the patient be unaware that he has had an attack. Running a short distance may be another manifestation of these attacks (precursive epilepsy), or there may be a few slight convulsive movements.

(3) *Psychic Epilepsy.* These attacks may precede a convulsion but usually follow either it or an attack of petit mal.

The symptoms may consist of maniacal excitement in which deeds of violence are done, or in which for a greater or less period of time the patient is apparently normal, may travel and perform other logical and reasonable acts, all of which he has no recollection of after the attack passes off. A similar state may occur in hysteria.

MYOCLONUS EPILEPSY.—Myoclonus epilepsy is characterized by convulsions of the ordinary type in the intervals between which there are myoclonic spasms either of mild or severe type (p. 766). There is a familial form of this disease.

Mental deterioration, depending in degree and rapidity of development upon the age of onset and frequency and severity of the attacks, usually occurs. There are epileptics, however, in which this is either very slight or does not occur.

Conditions to Be Differentiated from Epilepsy

Epilepsy must be distinguished from:

Syncope
Vertigo
Hysteria
Psychasthenia
Narcolepsy.

SYNCOPE.

Attacks of petit mal may resemble this. In syncope there is great weakness of the heart's action, sweating, and intense pallor. The attacks usually are due to some exciting cause[1] and are not periodical. The loss and return of consciousness are more gradual than in epilepsy.

VERTIGO.

Vertigo of the Ménière type may be mistaken, as the patient may fall and is confused. There is, however, rarely unconsciousness; tinnitus aurium is present; giddiness is pronounced and prolonged; and the signs of disease of the internal ear will be present (pp. 584, 602).

HYSTERIA.

Hysterical convulsions may be confounded and the two may coexist in the same patient.

The table on opposite page modified from Gowers gives the distinguishing features of each.

Conditions of *automatism* due to epilepsy are distinguished from those of *hysteria* by the history of previous epileptic seizures. If due to hysteria, other symptoms of hysteria will probably be or have been present. Various other hysterical stigmata may be present in epilepsy.

PSYCHASTHENIA.

Seizures somewhat resembling epilepsy sometimes occur in epilepsy. Consciousness is not completely lost and other symptoms (p. 795) are characteristic.

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[1] Epileptic seizures may also be due to an exciting cause, as trauma, or mental shock. The epileptic, however, will have a history of previous attacks occurring without apparent cause.

	Epilepsy	Hysteria
Apparent Cause	None	Emotion
Warning	May be an aura or there may be no warning	Palpitation malaise, choking, feelings of nervousness
Onset	Always sudden	Often gradual
Scream	At onset	During course of attack
Conciouness	Lost	Practically never entirely lost. Patient can be aroused and after attack can tell about what happened during attack
Convulsion	Tonic, followed by clonic spasms. Rarely tonic alone	Usually tonic spasm; if clonic movements are slow and consist of struggling, throwing the limbs about or arching the back
Biting	The tongue	Never the tongue; may the lips, hands, other people or things
Micturition	Frequent	Never
Defecation	Occasional	Never
Talking	Never	Frequent
Duration	A few minutes	10 min. to 1 hr. or more
Restraint Necessary	To prevent patient injuring himself	To control violence of patient.
Pupils	Dilated, not responsive to light	Responsive to light usually
Facial Expression	Pallor followed by lividity	Face may be pale, often flushed; may be no change
Termination	Spontaneous	Spontaneous or induced by water or apomorphin
Babinski Sign	May be present after a convulsion	Not present

Narcolepsy.

Petit mal may be mistaken for narcolepsy (See p. 799).

The spasms of tetany, tetanus, hydrophobia, and strychnin poisoning are not accompanied by loss of consciousness.

Care must be taken in all cases of epileptiform convulsions to determine if any of the causes mentioned above are present. If seen during the first attack and the patient is a woman, the possible existence of pregnancy must be inquired into.

4. Migraine

Migraine, or *hemicrania, sick headache,* or *megrim,* is a constitutional neurosis, associated with paroxysmal attacks of headache, usually confined to one side and accompanied by nausea, vomiting, other sensory and motor disturbances, vasomotor symptoms, and mental depression.

Occurrence.—It usually makes its appearence early in life, rarely beginning after thirty.

Predisposing Factors.—Heredity, either direct or indirect, is an important predisposing factor. If direct, members of the family for generations back have been affected. If indirect, they have been sufferers from other neuroses, as epilepsy or neuralgia. A gouty diathesis or auto-intoxication of some nature has an important influence.

Causes.—Syphilis has seemed to be a cause in a few cases (Collins). Fatigue, eye strain, excitement, or digestive disturbances may excite an attack.

Symptoms.—For a day or so before an attack there may be prodromes, as lassitude and an excessive desire for sleep. Often without warning pain will appear in either the eye, supra-orbital or temporal region of one side, which may soon involve one side of the head. Rarely both sides may be affected.

The pain is aggravated by noise, light or stooping, and is relieved somewhat by the recumbent position. The face is usually pale, and the extremities cold (angiospastic type).

In some cases, before the pain is felt, bright spots may be seen in one side of the visual field, or bright zigzag lines are seen. In rare instances other forms of visual hallucinations may be present.

After the pain has lasted for a number of hours, nausea followed by bilious vomiting occurs, and the pain disappears. Rarely instead of pallor, the face is flushed and there may be unilateral sweating (angioparalytic type).

The above is a description of an ordinary attack. In some cases, in addition to these symptoms there may be various paralytic symptoms, as lateral homonymous hemianopsia, aphasia, hemiplegia, hemianesthesia, monoplegia, and paralysis of cranial nerves.

These symptoms correspond to the side on which the pain is located; thus if aphasia occurred there would be left-sided pain, if there were right-sided hemiplegia there would be left-sided pain. They are due probably to arterial spasm and are usually transient. Often repeated attacks may become permanent (Hunt, Am. J. Med. Sci., Sept., 1915, p. 313).

Ophthalmic Migraine.—A form of migraine associated with paralysis of the motor nerves of the eyeball and also sometimes associated with mental depression and temporary loss of memory has been termed ophthalmoplegic migraine.

Conditions to Be Differentiated from Migraine

Migraine must be differentiated from:

Headache due to other causes
Neuralgia
Transient paralysis without headache
Ocular crises of tabes dorsalis.

HEADACHE DUE TO OTHER CAUSES.

Headaches from other causes (p. 570) differ from migraine in that they are not periodical, not associated with the peculiar vasomotor and paralytic symptoms above described, and a cause can usually be found.

NEURALGIA.

In neuralgia the pain is confined to nerve trunks, is either constant with shooting exacerbations of pain which follow the course of the nerves, or there is shooting pain with intervals in which pain is absent. The vasomotor and paralytic symptoms are absent.

TRANSIENT PARALYSIS WITHOUT HEADACHE.

Arterial spasm may cause apoplectiform attacks with transient paralysis (p. 713). In these attacks there is no headache. Epilepsy and migraine may sometimes coexist.

OCULAR CRISES OF TABES DORSALIS.

These may simulate migraine associated with visual hallucinations. Other symptoms of tabes would be present (p. 754).

5. Psychoneuroses

These are conditions in which the physical symptoms are more or less dominated by a morbid mental state. They consist of hysteria, neurasthenia, psychasthenia, the anxiety neurosis, and tics.

Hypochondria and dipsomania may also be included, but these are more purely mental states in which physical symptoms do not occur. Therefore they will not be described here.

Etiology.—There is underlying these conditions a congenitally unstable, nervous system. This may be manifested by nervousness, excitability, irritability, a tendency to worry without due cause, and to hunt trouble. Many of these people never develop other symptoms and are able to perform their daily tasks; but to do so requires more effort than does the normal person. Others, after some exciting cause either develop some form of insanity or one of the conditions mentioned above.

That similar exciting causes will cause one of these conditions to develop in some people and not in others shows that there must be a predisposition in those who do. This may be derived from a neuropathic or psychopathic ancestry, one in whom other similar conditions or insanity have existed, or from alcoholism, tuberculosis, and similar conditions in one or both parents.

(*a*) *Hysteria*

General Consideration.—Hysteria is the most common of these conditions. Of late years different observers have taught that certain groups of symptoms previously classified as hysterical, should not be so considered. As yet there is not unanimity as to which these symptoms should be, and in the following description the old views will be followed. The symptoms of what are now termed the *anxiety neurosis* and *psychasthenia*, respectively, will be mentioned, as these terms are in frequent use in the literature.

It is not possible in the space at command, nor does it seem necessary, to detail the many views as to what is and what is not hysteria, and the psychogenesis of the symptoms.

It is important, however, to remember the following:

(1) The person liable to hysteria is characterized by a constitution, often congenital, in which there is an excessive reaction to emotional stimuli which are often in themselves trivial. Emotional traumatism of some sort is the exciting cause of hysteria.

(2) This leads to a condition in which there is hypersuggestibility, the suggestion being either generated within the mind of the patient or received from without. In this way, to a large extent at least, the symptoms are induced and, it may be said also, removed. Babinski has defined hysteria as a condition in which the symptoms are induced by suggestion and removed by persuasion. There must be an abnormal mental condition, as stated above, to bring this condition about; it is not present in a normal person.[1]

(3) The symptoms themselves are probably, as stated by Janet, due to a dissociation of the personality. The synthesis of mental processes and ideas into a coherent whole constitutes the personality or ego.

This constitutes the condition of waking, or normal consciousness. We have also a condition of subconsciousness, in which groups of ideas exist that are not under ordinary circumstances recognized, yet they may produce results.

The hysterical process may cut the psychic representation of a limb out of the field of consciousness, and motor or sensory paralysis results. In other words, there is a defect in attention so that sensations from the arm are not perceived or motor impulses are not sent from the motor centers, as the case may be.

It may act in another way, the personality at one time being guided by one set of ideas and at another time by a different set, as those of the subconscious mind. Memory of what takes place when governed by one set being absent when under the influence of the other. States of double personality, somnambulism, and trance states are so caused.

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[1] All normal persons are somewhat liable to receive suggestions, but they also can fight them and oppose them.

The definition of Crocq, that hysteria is "a psychopathological state characterized by hyperimpressionability, diminution of cerebral control, and hypersuggestibility," covers the ground fairly well.

The views of Freud, while not largely adopted, may be mentioned. He believes that the emotional trauma is practically always of a sexual nature, although not necessarily sexual. They may have to do with modesty, parental and filial affection, and all sexual experiences and perversions. The trauma has usually occurred before the age of puberty, but through shame or disgust, they are buried in the subconscious mind, and hence not recognized by the conscious mind. The effort of these episodes to come into consciousness and the effort to repress them causes a conflict, and hence the production of various symptoms which have the value of a compromise between two psychic streams.

Causes.—As has been stated above, *heredity* is the *predisposing cause.* In about seventy-five per cent of the cases there is a history of hysteria or some neurosis or psychosis in one or both parents.

The most important *exciting cause* is emotional disturbance which may be due either to fright, either associated with or without physical trauma, excitement, especially religious, sorrow or anxiety. Various toxemiae, as alcohol, lead, mercury, tobacco, and the infectious fevers, may excite it. Mental, physical, and sexual excesses may also be factors, and it has been developed by imitation.

Occurrence.—It may develop at any age—most cases occurring between fifteen and twenty-five in females, but later in males. It is more common in females, and in the Jewish and Latin races. It is not uncommon in negroes.

Symptoms.—The symptoms vary in severity. A large number of cases, as seen in this country, are often described as suffering from nervousness.

The *mental attitude* of *all* hysterics consists of a more or less intense desire for notoriety and sympathy, to secure which they will often lie, exaggerate, and deceive. Self-mutilation and injury may be practiced.

In addition to this mental state there may be no other symptoms than lack of emotional control, shown by outbursts either of weeping or laughing or both alternately, without adequate cause. This may be due also to organic disease, as in bulbar palsy, multiple sclerosis and disease of the optic thalamus and corpus striatum. Other symptoms of such diseases will of course be present.

Preceding these attacks there may be a feeling of fullness or choking in the throat (globus hystericus). Irritability, mental depression, undue sensitiveness, headache which may be localized in one spot (clavus hystericus), spells of trembling and vague pains in the back, ovarian or cardiac regions, may be present. In some cases attacks of somnambulism may occur. Such symptoms go to make up the condition sometimes termed *hysteria minor.*

Hysteria major consists of the above mental state and general symptoms plus various forms of crises or paroxysms and more or less permanent or interparoxysmal symptoms.

The *crises* may consist of either emotional outbursts, as above described, paroxysms of pain simulating angina pectoris and gastralgia, nausea and vomiting, prolonged coughing, hiccoughing, sneezing and rapid breathing, trance, lethargy, catalepsy, cerebral automatism, or convulsions. Many of the above may also be interparoxysmal symptoms and will be described further on.

Convulsions, as described by the French authors, are not common in this country. As seen here, the patient after some exciting cause (*supra*) may either have an emotional outburst, as described above, and then fall, or the attack develops suddenly without prodromes.

After the fall, which is done so that injury does not occur, the patient becomes either rigid or the limbs may be moved about in more or less purposive movements. Consciousness is not lost, but may be apparently so. After it is over patients will often say that they knew what was going on about them but were powerless to answer questions.

Still milder attacks may occur consisting of rapid clonic spasms resembling very much a hard chill and without even apparent disturbance of consciousness.

In the *severe attacks* muscular rigidity is marked and may be followed by slow muscular contractions, causing the body to be bent in various directions, usually in opisthotonos. The face may be red and some saliva exude from the mouth. This may be followed by clonic spasms, or the patient may assume various attitudes and emotional states. A delirious condition may be present in which the patient talks continually or the cries of animals may be imitated.

These attacks may last from five or ten minutes to an hour or more. After it ceases, the patient may be either apparently normal, show some of the interparoxysmal symptoms, or pass into a condition of lethargy, trance, somnambulism or automatism, the so-called hysterical dream states.

These conditions may also occur independently or be followed by convulsive seizures. In a state of *lethargy,* the patient lies in a semistupor with closed eyes, the lids of which often show fine fibrillary tremors. *Trance* is an intensification of lethargy. Food is apparently not taken, and while the weight may diminish, temperature, pulse and respiration do not seem to be much affected. A *cataleptic condition* may also occur in which the limbs are rigid and remain in any position in which they are placed (Fig. 189). These states may continue for weeks.

Attacks of *somnambulism* may occur, or the patient may attend to his daily affairs and even travel and perform other purposive and proper actions and, on coming to himself, have either a clouded or no recollection of what he had been doing—automatism. During these states criminal acts may be committed. More or less, retrograde amnesia is present in all of the above states.

In double personality the patient may possess one personality for a time, then suddenly change to another, the character of which may be just the opposite of the first, as a Dr. Jekyll and Mr. Hyde. The acts performed by the first personality are not remembered by the second, but may be revived by putting him in the hypnotic state.

Another peculiar mental state is the so-called *Ganser's syndrome.* In this, the patients act foolishly, as if they were children; they give short, snappy and foolish answers, laugh without cause, and imitate the cries of animals. This condition may also occur in alcoholism, epilepsy, and dementia precox.

The *interparoxysmal symptoms, also* called the *stigmata,* may be divided into *sensory, motor* and *visceral.*

Sensory Symptoms.—Sensation may be diminished, lost, increased (pain and hyperesthesia) and perverted (paresthesia).

Some form of sensory loss or diminution is common. It may involve either the entire person, one-half (hemianesthesia), or occur in scattered patches (Fig. 186), or in segmental form resembling a glove or stocking (Fig. 187).

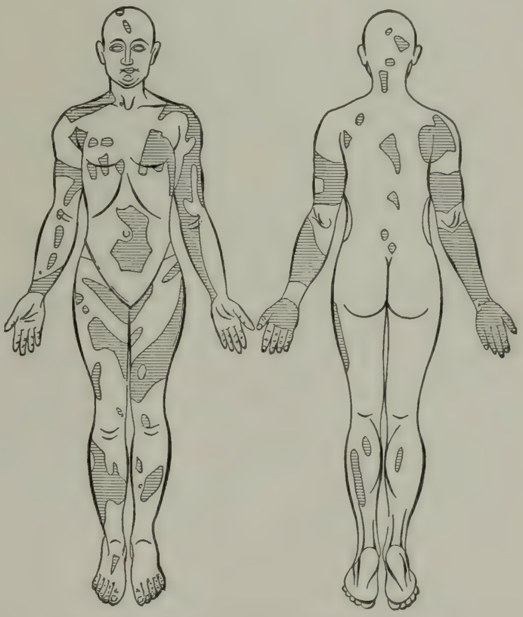

Fig. 186.—Horizontal Lines Indicate Areas of Anesthesia. (After Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

All forms, i. e., touch, pain, temperature, muscle, may be affected, or only one or more. According to Pitres, touch is never lost alone; pain sense frequently is.

Allochiria (p. 567) is a symptom characteristic of hysteria. Other peculiar phenomena are the *phrictopathic sensations.* These consist of either abnormal persistence, the sensation persisting for a number of seconds after the stimulus is removed; delay in perceiving the sensation (this also occurs in organic disease, notably tabes); a sudden motor response resembling a true reflex; a sensation as if the part stimulated did not belong to the patient, or a disagreeable or paresthetic sensation no matter what the nature of the stimulus.

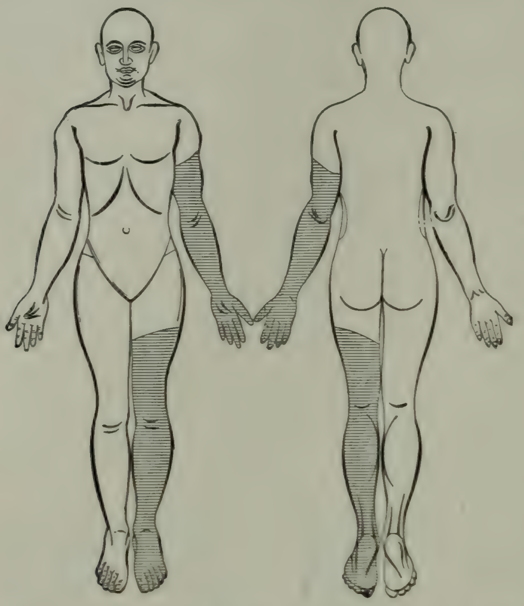

Fig. 187.—Horizontal Lines Indicate Areas of Anesthesia. (After Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

The skin reflexes in the anesthetic area are sometimes but not always absent or diminished.

The special senses frequently suffer, that of sight most commonly so; it may be either completely lost or partially so. In the former, development is often sudden and the recovery equally so. It is sometimes unilateral. The latter is shown by either contraction of the visual fields, loss of or diminished color perception (p. 600), tubular fields, in which the contracted field remains the same size no matter at what distance the point of fixation is placed; oscillating fields, in which the test object appears and reappears several times in the same meridian; the shifting or exhaustion field, in which it is constantly changing during the examination (this also occurs in neurasthenia), and homonymous hemianopsia (very rare).

Deafness is usually present, if at all, as part of a hemianesthesia and may respond to the tests for deafness of nerve origin (p. 602).

Smell and taste also are usually impaired unilaterally and are part of a hemianesthesia (p. 602).

Hyperesthesia is the cause of the so-called *hysterogenic zones.* These are localized hypersensitive areas, light pressure upon which will often excite a paroxysm and also cause one to cease after it has developed. They are most commonly found over the ovaries, under the breasts, along the spine, and on the trunk beneath the ribs. Retinal hyperesthesia may occur with dread of light, blepharospasm, and lacrimation (p. 754).

These and other areas may be the seat of spontaneous pain (topalgia) and true inflammation simulated. This may occur in the joints, abdomen, and cardiac regions. When in the abdomen, appendicitis and other abdominal affections have been mistaken; when in the cardiac region, angina pectoris may be simulated (p. 430).

Paresthesia, if present, takes the form of either flashes of heat and cold, crawling sensations, sensations of numbness, etc.

Motor Symptoms.—The motor symptoms consist of paralysis, contracture, tremor, and incoördination.

Paralysis may take the form either of monoplegia, hemiplegia, paraplegia, diplegia, or be limited to a few muscle groups. It may follow a convulsion or develop independently of this, either gradually or suddenly, after one of the exciting causes mentioned (p. 775).

The *degree of paralysis* varies from slight weakness to complete loss of power. The deep reflexes are either increased, normal, diminished, or lost; the latter two are rare. True ankle clonus does not occur, but a pseudoclonus, in which the movements are not so persistent as in true clonus, does. Contracture may or may not coexist.

Local paralysis is more rare. It may affect a group of muscles of a limb or those of the eye, larynx, pharynx, and esophagus. Paralysis of the *eye muscles,* either intrinsic or extrinsic, is rare but may occur. A curious symptom is *monocular diplopia,* or polyopia, in which one eye sees two images. It may be due also to corneal deformity, and is then not hysterical. Apparent ptosis may occur. Pseudoparalysis due to spasm of these muscles is more common. The diagnosis may be difficult, and requires an expert ophthalmologist.

The *vocal cords* are often paralyzed, causing either complete aphonia, or a whispering voice (p. 650). It may appear and disappear suddenly. The adductors are always the muscles affected. Paralysis of these muscles may also be due to local inflammation or overuse of the voice. In hysteria anesthesia of the mucous membrane frequently coexists. In paralysis of the pharynx and esophagus there is difficulty in swallowing. Examination with a bougie will fail to show evidence of stricture, and the x-ray will probably show a tumor in the mediastinum; other symptoms characteristic of that condition would also be present.

Paralysis of *other cranial nerves* is exceedingly rare. Pseudoparalysis due to spasm is more common.

Contractures may attack any group of muscles and may be of either the monoplegic, hemiplegic, or paraplegic type.

They usually develop gradually, may persist for years, and then disappear suddenly. If long continued there may be some wasting of the muscles from disease and some adhesions in the joints. Unless these are marked, the contracture relaxes under an anesthetic. A common type is shown in Figure 188.

Pseudo-abdominal tumors and *false pregnancy* (pseudocyesis) are caused by spasm of the diaphragm associated with relaxation of the abdominal muscles and distention of the intestines with gas.

Tonic spasm of the muscles of the face, tongue, eyes, and neck causes deformities similar to those caused by paralysis of the same muscles, such as may occur in organic hemiplegia. Instead, however, of the angle of the mouth being drawn away from the affected side it would be drawn to it and the tongue when protruded, instead of going toward the affected side, would go away from it.

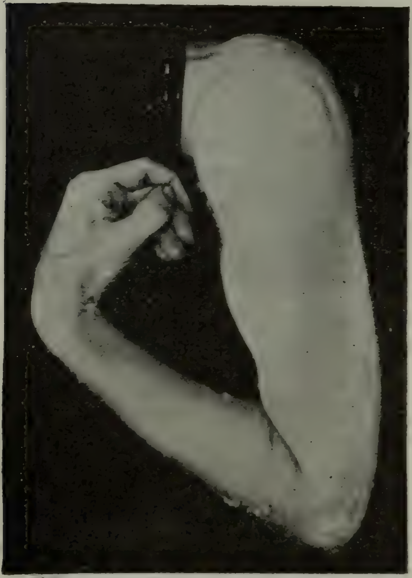

Fig. 188. — Hysterical Contracture. (Methodist Episcopal Hospital, Philadelphia; from Potts' "Nervous and Mental Diseases," published by Lea and Febiger, Philadelphia.)

Clonic spasms of the muscles of the trunk and limbs may occur, causing peculiar movements (p. 766), or if milder in type, an apparent chorea. The movements are quicker and more coördinate than in true chorea.

Peculiar clicking noises may be caused by clonic spasm of the soft palate and pharyngeal muscles. Persistant hiccough is due to spasm of the diaphragm.

Tremor of any type may occur. They are often slow and coarse and may be of the intention type.

A peculiar form of *incoördination* may occur in which, while at rest, the patient may be able to move the limbs normally, but is unable either to stand or walk, or else does so in a peculiar way which does not resemble the gait of any organic disease. This is known as *astasia-abasia.*

In another condition, known as *akinesia algera,* every movement is associated with intense pain, although there is no evidence of inflammation of muscles or joints.

Visceral Symptoms.—The visceral symptoms are many.

Respiratory Apparatus.—Those referred to the respiratory apparatus are apparent dyspnea, without distress or interference with the pulse rate; irregular rapid or slow breathing; hiccough; peculiar sounds either with inspiration or expiration, gaping, yawning, sneezing; cough, which is dry, persistent, brazen, and paroxysmal.

Blood which comes from the mouth or pharynx may be expectorated. It must not be mistaken for true hemoptysis. Examination of the sputum will distinguish the two.

Digestive System.—Depraved and perverted appetite is common in hysteria. In the so-called *anorexia nervosa* there is complete loss of appetite and refusal of food; this may be associated with regurgitation or vomiting, if any is taken. Great emaciation occurs and death may result

(See p. 284). Either diarrhea—especially after the taking of food, constipation, painful spasm of the anus, or fecal vomiting, may occur.

Cardiovascular System.—Symptoms referable to the cardiovascular system are either rapid or slow pulse, severe palpitation, and pain simulating that of angina pectoris. It rarely extends down the arm and does not cause rapidity of the heart's action and the intense anxiety, as does true angina.

Vasomotor Symptoms.—Vasomotor symptoms, as angioneurotic edema, dermographia, urticaria, flushings, pallor, and edema, which may be either of a pale, waxy character, pitting upon pressure, or of a bluish tinge which does not pit. In the latter the temperature of the affected limb is below normal.

Cases of hysterical fever have been reported. It is rare. The diagnosis depends on the absence of positive symptoms referable to any known cause of fever, the retention of nutrition, and the possible presence of other stigmata of hysteria.

Conditions to Be Differentiated from Hysteria

Hysteria must be distinguished from:

Neurasthenia
Psychasthenia
Anxiety neurosis
Hypochondria
Dementia precox
Malingering
Various forms of organic disease of the nervous system and viscera which it may simulate.

NEURASTHENIA.

Neurasthenia may coexist with hysteria. The table on page 782, by Ziehen, gives the essential differences:

PSYCHASTHENIA.

In psychasthenia there are obsessions and fixed ideas which dominate the actions of the patient. In hysteria, they are not apparent and do not dominate his action but do his body and functions. As Dana puts it, the obsession in hysteria is a subconscious one; in psychasthenia, it is conscious.

ANXIETY NEUROSIS.

The peculiar characteristics of the anxiety neurosis are described on page 796.

HYPOCHONDRIA.

Hypochondria is more likely to be confounded with neurasthenia, but as the hypochondriac believes he is ill and constantly talks about his

	Hysteria	Neurasthenia
Sensibility	Mostly unilateral, patchlike or regional anesthesia, hypesthesia, hyperesthesia and analgesia and hyperalgesia	Intact or generally increased
Pressure Points	With hysterogenic, and eventually also hysterophrenic characters, more marked; mostly on one side; usually combined with hyperesthesia	Without such characters, usually symmetrical, and with intact skin sensibility
Paralysis	Not unusual	Nearly always abnormal tire
Headache	In localized spots, rarely a sense of pressure	Often bandlike and sense of pressure frequent
Visual Fields	Diminished	Only diminished under influence of fatigue
Smell, Taste and Hearing	Often involved	Involved symmetrically, and usually as a hyperesthesia
Skin Reflexes	Often unequally modified	Rarely different on two sides of the body
Attacks	Often typical	None, or rarely attacklike emotional movements set up by affectation; mostly depressive ideas
Mood	Excessively variable	Irritable or hypochondriacal
Intellectual Activity	Disturbed, especially the attention	Disturbed by reason of early fatigue
Suggestibility	Marked	Influence sight
Sleep	Often excellent	Mostly bad
Course	Polymorphous	Rarely polymorphous

symptoms, and hysteriacs also desire to draw attention to themselves and hence to complain of various symptoms there may be confusion. In hysteria, the mind of the patient will dwell on other things; he is not so depressed and actual objective symptoms will usually be found. Furthermore, the symptoms may often be made to disappear by suggestion.

DEMENTIA PRECOX.

Dementia precox in its early stages may present symptoms of a hysterical nature (See p. 777). Evidences of mental failure will be found, and sooner or later, delusions will be manifest which will govern the patient's behavior.

MALINGERING.

Malingering will be discussed under the traumatic neuroses (p. 794).

ORGANIC DISEASE OF NERVOUS SYSTEM AND VISCERA.

Hysteria may simulate disease of any organ. At the same time there are certain peculiarities about the symptoms which usually render a diagnosis possible. The symptoms are apt to change from day to day, especially under the influence of suggestion. There is also always something that is different from similar symptoms due to organic disease. The mental state of the hysterical and the history of the manner of onset and possible exciting cause must always be taken into account (p. 775; see also Vagotonia, p. 596).

In this connection, it must be borne in mind that hysteria frequently coexists with other diseases. Therefore even if undoubted symptoms of hysteria are present all possibility of other disease being also present must be eliminated before a diagnosis of pure hysteria is made.

The following peculiarities of symptoms simulating those due to other causes will aid in the distinction.

In *hysterical somnambulism* the condition usually follows a paroxysm or convulsive seizure. In *true somnambulism,* the attack occurs at night either without apparent cause or following a heavy meal, mental strain or excitement. They occur more or less periodically and the patient may be apparently normal between the attacks. Both, however, occur in neuropathic persons.

Catalepsy must be distinguished from that occurring in *dementia precox.* The latter type is probably more common. The diagnosis must depend on the history of the patient previous to the development of the symptoms and possibly the symptoms occurring after the condition disappears.

Epileptic automatism usually follows an epileptic seizure (See p. 769); *hysterical automatism* does not. Neither will there be a history of previous epileptic seizures, although there may of hysterical ones and the presence of hysterical stigmata.

Hysterical edema may be mistaken for *angioneurotic edema.* The points of difference are given on page 809.

Loss or diminution of sensation due to hysteria does not follow the distribution of either peripheral nerves or segments of the spinal cord (Fig. 123). The peculiar forms shown in Figures 186 and 187 do not occur in organic disease.

If motor paralysis is present, the sensory paralysis does not correspond in its distribution to that of the motor. It varies in distribution from day to day, or even during the examination, and may be made to disappear by suggestion, which may be increased by the use of electricity or magnets, or by the application of coins or pieces of metal to the anesthetic area. If the skin is pricked in the anesthetic area the blood may either not flow at all, or does not flow as freely as in anesthesia due to organic causes. Hypesthesia or hypalgesia are more common than anesthesia or analgesia.

Loss of temperature sense may occur but loss of deep muscle sense is rare.

If hemianesthesia is present it is usually on the left side and is complete, involving the skin, mucous membranes, special senses, and often the muscles and joints. The line of demarcation at the middle line is much sharper than in organic hemianesthesia. In fact, the line of demarcation between abnormal and normal sensation is always sharp in hysteria. In organic disease they shade into each other more or less gradually, and if a hemianesthesia, is not nearly so complete as above described.

In hysterical hemianesthesia irritation of the nasal mucous membrane will cause a flow of tears even if the irritation is not felt. If due to any organic lesion the reflex will be absent and the tears will not flow.

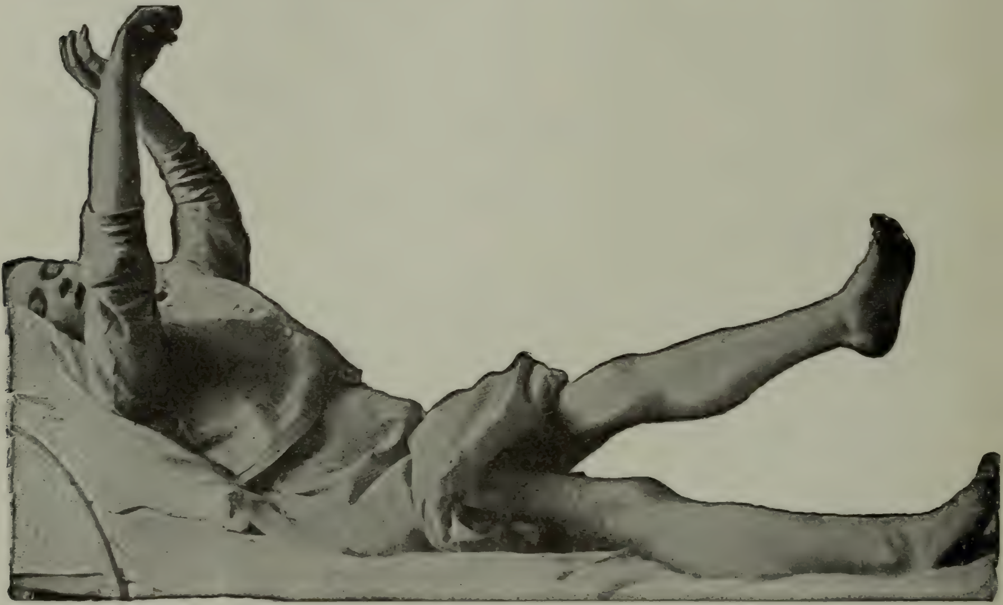

Fig. 189.—Hysterical Attack with Catalepsy. (After Strümpell.)

The patient with hysterical anesthesia usually is unaware of its existence, as he does not experience numbness. It may be said here that the patient actually feels, but his mind is unable to recognize it.

The same is true of the special senses. A persistent homonymous hemianopsia associated with hemianesthesia is in favor of an organic lesion. Ophthalmological and otological examination will uncover *hysterical blindness and deafness,* showing the optic nerve to be normal, and while some of the tests (bone conduction) of nerve deafness may be positive, all of them are not.

Hyperesthetic areas may be mistaken for pain due to visceral disease, appendicitis, cholecystitis, etc.

In this connection the researches of Head (p. 567) must be borne in mind. Careful examination by all the means known to internal medicine must be made, remembering that hysteria may be present in addition to some visceral disease. If hysterical, pressure may cause a convulsive paroxysm and of course other hysterical stigmata must be looked for.

Motor Paralysis.—Motor paralysis may present some difficulty, especially *paraplegia* associated with loss of sensation.

In the hysterical form the Babinski reflex is not present, incontinence of urine and feces is rarely if ever present, although retention may be, and the distribution of the anesthesia does not correspond to the distribution of the motor paralysis. For instance, hemianesthesia may be associated with paraplegia, or while motor paralysis is complete in both legs the anesthesia only extends to the knees. Excepting possibly a slight amount due to disease, muscular atrophy does not occur in hysteria.

In *hysterical hemiplegia* the history of development is usually different from that of the organic type. It often follows an injury to the affected side. The leg is usually more affected than the arm, which is the opposite of what usually occurs in organic paralysis. The Babinski reflex will be absent; paralysis of the face is exceedingly rare—spasm sometimes causing an apparent crossed paralysis is more common. The leg is dragged or pushed along, not swung as in organic hemiplegia, and the foot held in unusual positions (Compare Fig. 190 with Fig. 177).

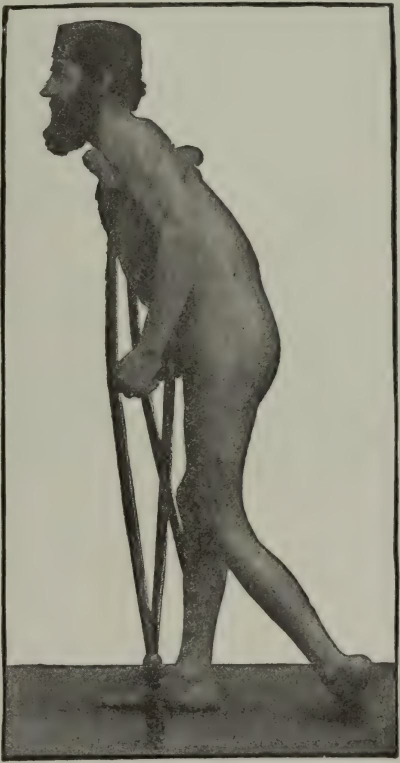

Fig. 190.—Hysterical Paralysis of the Leg. (Icon de la Salpêtrière.)

Tests.—The following tests may be applied to determine the question:

Babinski's "Combined Flexion of the Hip and Trunk."—This is sometimes called Babinski's "second sign," and consists in making the patient lie on his back on a hard surface, with the arms folded and the legs separated. If in this position he attempts to sit up without using his arms, the leg, if affected with hysterical paralysis, will *remain flat* on the table or bed, while the other limb will rise slightly. If due to organic paralysis, the affected leg will *rise higher* than the normal. In healthy persons both legs rise equally from the surface. If after sitting up the patient (his arms folded) lies down again similar phenomena occur.

Hertz has found that cases of hemichorea, when similarly tested, will act similarly to cases of organic paralysis.

Léri's Forearm Sign.—This consists in flexing the fingers of the patient on the palm, the latter on the forearm, rolling, so to speak, the hand in on itself with some force.

In a normal person, or in one with hysterical paralysis, while this is being done the forearm flexes on the arm. In organic paralysis it either does not do so at all or does so much less than does the other side.

A normal person, lying as in testing for the Babinski sign (*vide*), can elevate either leg or both together without bending the knees. If organic paralysis is present and he attempts to do this, there will be bending of the knee, provided there is some power (Noica and Paulan).

Grasset and Gaussel Test.—Another test, useful if the *paralysis is not complete,* is that of Grasset and Gaussel. It consists in the inability of a patient with organic hemiplegia while lying down to raise both legs simultaneously from the surface, while he can raise either leg separately.

Another method of making this test is to have the patient raise the leg of the paralyzed side; while it is held in the air the sound leg is raised up, when the paralyzed leg will at once fall. In hysteria there is no difference whether the legs are raised simultaneously or separately.

Hoover's Test.—Hoover's test is based on the fact that if a normal person, while lying on his back, elevates one leg, the heel of the other is pressed downward with equal force. This can be felt by placing the hand under the heel while the leg is being raised.

If in organic paralysis of the leg the effort is made to raise it, the heel of the sound limb is similarly pressed downward. If, however, the sound leg is raised, the amount of downward pressure of the paralyzed limb will be proportionate to the amount of weakness—none occurring if the paralysis is complete. In either hysterical or simulated paralysis the opposite occurs.

Attempts to move the paralyzed leg do not cause downward pressure of the sound leg, but if the sound limb is raised complemental opposition occurs in the paralyzed limb. If able to walk it will be noticed that in either organic hemiplegia or crural monoplegia the patient can walk sideways well toward the paralyzed side, but badly toward the sound side. In hysterical cases moving sideways is badly performed in either direction.

Beevor states that in *functional paralysis* the antagonistic muscles (p. 564) do not relax when the active muscles contract—in fact they may contract first, which causes the to-and-fro or tremulous movement often seen when attempts at movement are made in these cases.

Oppenheim has described two signs of organic hemiplegia useful when there is but slight weakness. *The orbiculopalpebral sign* is elicited by causing the patient to close both eyes firmly. The examiner then attempts to open the eye by separating the lids. If slight paralysis is present it will be found easier to open the eye on that side. If conjunctivitis is present this sign is not reliable.

The orbiculolabial sign is shown by causing the patient to close his mouth tightly. The examiner tries to separate first one corner, then the other. The resistance is less on the paralyzed side.

Mingazzini describes the following: The patient is made to hold out both arms straight in front of him with his eyes shut; after a minute or so the arm on the affected side tends to drop or show evidences of fatigue sooner than the other. The same will occur if, while lying on the back, both legs are elevated to an angle of about forty-five degrees.

Contractures may be mistaken for either spasticity or deformity due to organic disease. In hysteria muscles are frequently affected and deformities caused that never occur in organic disease (See also p. 558). The contractures due to central neuron disease (p. 563) are due to overaction of the flexors; in hysteria extensors may suffer most (Compare Fig. 188 with Fig. 112).

In deformities due to the overaction of antagonistic muscles, as in poliomyelitis, there will be atrophy, reactions of degeneration, etc., in the weakened group of muscles.

Astasia-abasia may be mistaken either for ataxia or motor paralysis. The essential feature is that the difficulty in using the limbs only appears when attempts are made to stand or walk; when at rest they can be and are moved about normally.

The following organic diseases are especially liable to be mistaken:

Multiple sclerosis
Cerebrospinal syphilis
Chorea
Epilepsy
Organic hemiplegia
Transverse myelitis.

MULTIPLE SCLEROSIS.

In multiple sclerosis the tremor and spasticity may resemble that of hysteria. It must be remembered also that they may both be present in the same patient. Ankle clonus is common in the former, but rare in the latter. The Babinski reflex does not occur in hysteria (it may be absent sometimes in multiple sclerosis). Optic atrophy, nystagmus, the characteristic speech disturbances, well marked palsy of the ocular muscles or of others supplied by cranial nerves, do not occur in hysteria. Diplopia may sometimes, but it does not increase upon lateral movements of the eyes (p. 601).

Attention to the tests and symptoms mentioned above may aid in distinguishing organic from functional paralysis.

CEREBROSPINAL SYPHILIS.

In cerebrospinal syphilis there may be optic neuritis; there is often intense headache—worse at night (hysterical headache is usually better). Paralysis is differentiated by the methods given above. Serological tests will show the presence of syphilis (p. 758).

CHOREA.

With chorea, hysteria may also be present. The movements of chorea are more diffuse; that is, there is a greater variety of movement and more muscles are affected. In hysteria there is apt to be a repetition of the same movement and the contractions are sharper and quicker. Hysterical chorea may develop by imitating the movements of a genuine case.

EPILEPSY.

The distinction from epilepsy is given on page 771.

ORGANIC HEMIPLEGIA AND TRANSVERSE MYELITIS.

The differences between functional and organic paralysis are given on page 785.

The affections of the joints and the various visceral diseases simulated must be distinguished by careful attention to all methods of diagnosis, especially laboratory and x-ray.

(*b*) *Neurasthenia*

Neurasthenia is a condition usually known to the laity as "nervous prostration."

Characteristic Features.—It is characterized by an abnormal liability to fatigue, either mental, physical, or both combined. In addition to the functional weakness there is an increased irritability of the nervous system. The symptoms, therefore, are due to nervous weakness and nervous irritability.

Causes.—The underlying cause is a neuropathic constitution, the causes of which are detailed on page 773. The exciting causes are overwork, both mental and physical; prolonged physical or mental strain; physical or mental shock or injury; the excessive use of alcohol, tea, coffee, tobacco; infectious diseases, especially influenza and syphilis; autointoxication due to chronic constipation; intestinal indigestion; kinks and ptoses of the gastro-intestinal tract; sexual excesses; hyperthyroidism and other ductless gland disturbances. A relatively small proportion develop at the time of the menopause.

Dana has summarized the leading causes thus:

Hereditary nerve instability
Overwork and worry
Severe shocks, with or without injury
Infections
Abuse of stimulants and narcotics
Abuse of sexual functions
Disorder of digestive functions
Autotoxemia
Glandular disturbances.

Symptoms.—The symptoms may be divided into psychic, motor, sensory, and visceral.

Psychic Symptoms.—One of the most important of these is interference with sleep, as either insomnia, disturbed sleep due to disagreeable dreams, or inability to sleep sufficiently long.

Even if sleep is obtained it is not refreshing, and the usual complaint of the patient is that he is more tired after awaking than when he went to bed. There is inability to maintain prolonged intellectual effort, especially to keep the mind concentrated on one subject; there is also loss of confidence and ambition. Irritability of temper and emotionalism are prominent. There is marked introspection, the patient placing undue importance on the slightest symptom or peculiar sensation.

Motor Symptoms.—There is usually great liability to fatigue, the patient tiring after comparatively slight muscular exertion. There may be a fine tremor of the hands, sometimes of the eyelids when closed, and rarely, fibrillary twitchings of the muscles. The knee jerks are often increased, but may be either normal or decreased. Even when increased, they soon become exhausted if repeatedly tested.

Sensory Symptoms.—Headache is frequent and is described either as a sense of pressure on top of the head or as if a tight band were tied about the head. Backache is also common, usually in the lumbar region. There may also be burning sensations about the scapula. The spine may be tender in certain areas, especially the lower cervical, middorsal, and lumbrosacral regions. This differs from the tenderness due to organic vertebral disease, as it is more pronounced when light pressure is made (spinal irritation). It may exist when spontaneous pain is absent. Pains and aches in other parts of the body may also be complained of. Paresthesia, as numbness, tinglings, sensations of heat and cold, and tightness or pressure in various parts of the body, may be present. There is often a general feeling of tire and exhaustion.

The *special senses* may suffer. Asthenopic symptoms may appear after using the eyes. In such cases refractive errors must be excluded. Retinal hyperesthesia, shown by avoidance of light, profuse lacrimation and blepharospasm, when exposed to it, may be present.

Peculiar changes may occur in the visual fields; they have been termed "fatigue contractions." Thus when first tested it may be normal, but under repeated testing, contraction takes place in all or only certain meridians. After a rest it will become normal again, in that way differing from the contracted field of hysteria.

Another form is the fatigue or exhaustion spiral field, in which the points when united, form a tracing like a watch spring. Patients may complain that when they awaken in the morning they find it impossible to open the eyes unless they raise the eyelids with the fingers (night ptosis of Gowers). It may also occur if they awaken during the night (night ptosis of Weir Mitchell).

Muscæ volitantes, i. e., specks floating in front of the eyes, are often complained of. This symptom may also be due to certain organic diseases of the eye.

Tinnitus aurium may be complained of (p. 645).

Hyperesthesia of the auditory nerve, in which slight noises cause much discomfort, may occur. This might also be due to the general irritability of temper common to the condition.

Visceral Symptoms.—These consist of disturbances of digestion, circulation, secretion, and the sexual functions.

Digestive symptoms are common, and consist of hyperchlorhydria, lack of appetite, heaviness and fullness after eating, and flatulence. The tongue is often large, flabby, and marked with the teeth; it may or may not be coated. Constipation is the rule.

Circulatory Symptoms.—A common one is palpitation of the heart; persistent tachycardia is not uncommon. Pain in the cardiac region is sometimes complained of. Aortic pulsation, causing throbbing in the epigastrium and which in thin people can be easily seen, may be mistaken for aneurism. The throbbing in this is expansile, which is not the case in aortic pulsation.

Flushings of the face, urticaria, coldness of the hands and feet, may be present. The secretions—urine, saliva, sweat—may be either lessened or increased. The urine is frequently loaded with either oxalates, phosphates, or urates. Phosphatic urine is cloudy while being passed, often causing the patient to think he has spermatorrhea. Night sweats sometimes occur.

Sexual Symptoms.—In many cases these symptoms dominate the picture of the disease; so much so that they are termed "sexual neurasthenia." In men they consist of frequent nightly emissions, often without previous dream, premature ejaculation, incomplete erection, lack of sexual desire, and absence of the orgasm. Such cases often have chronic prostatitis, and the discharge of prostatic secretion causes much worry, as it is believed to be spermatic fluid. The above-mentioned disease, due to either frequent masturbation or sexual intercourse, coitus interruptus, or previous gonorrhea, may be the cause of the neurasthenic symptoms.

In women this form is not so common, but bad sexual habits, especially coitus interruptus, may have much etiological significance.

All of the symptoms mentioned above are not present in every case; in some mental symptoms predominate, in others, physical.

Conditions to Be Differentiated from Neurasthenia

Neurasthenia must be distinguished from:

Various visceral diseases
Hysteria
Hypochondria
Exophthalmic goiter
Vagotonia
Psychasthenia
Paretic dementia
Tabes dorsalis
Melancholia
Anxiety neurosis
Dementia precox.

Various Visceral Diseases.

Before making a diagnosis of neurasthenia it must be remembered that neurasthenic symptoms may either precede or accompany various visceral diseases. It is only necessary to mention these, their symptoms being found under their respective headings. This fact emphasizes the importance of careful examination.

The following must be considered: tuberculosis, anemia, gastric cancer, dilated stomach, ptoses and kinks of the gastro-intestinal tract, uterine and ovarian disease, pellagra, arteriosclerosis, syphilis, chronic prostatitis, and the possibility of chronic intoxications, as from lead.

Hysteria.

Hysteria is differentiated on page 782.

Hypochondria.

In hypochondria the fact of his having this or that disease is what dominates the mind of the patient and governs his actions. In neurasthenia, while the patient is introspective and watches his symptoms, the principal feature and cause of complaint is the inability to perform mental or physical effort owing to the fatigue so caused. In hypochondria the organs complained of will be found to functionate normally; this is not always so in neurasthenia.

EXOPHTHALMIC GOITER.

In exophthalmic goiter the patient is nervous, unable to exert himself, and the disease may follow fright. In abortive cases in which the classical symptoms are not marked there may be some difficulty.

Enlargement of the thyroid will, however, be found; the tremor is usually more marked and the tachycardia more extreme than is usually present in neurasthenia.

VAGOTONIA.

Disorders of the vegetative nervous system, as vagotonia, may be mistaken. Symptoms of and tests for these conditions are given on page 596.

PSYCHASTHENIA.

Symptoms peculiar to psychasthenia are given on page 795.

PARETIC DEMENTIA—TABES DORSALIS.

In the early stages of both paresis and tabes dorsalis, neurasthenic symptoms may be prominent and those complained of. Careful examination should cause the avoidance of error.

MELANCHOLIA.

Melancholia of a mild type may be mistaken. The mental depression is greater, the insomnia more pronounced. The complaint of undue fatigue is not made. The neurasthenic worries because he cannot work, the melancholic because he has done something for which he is or should be punished.

ANXIETY NEUROSIS.

The symptoms of anxiety neurosis are given on page 796.

DEMENTIA PRECOX.

In the early stages of dementia precox neurasthenic symptoms may be prominent. Here again the element of fatigue does not enter. If the patient with dementia precox refuses to work it is because he is not treated right or his merits are not recognized. Delusions and erratic behavior sooner or later appear.

(c) *Traumatic Neuroses*

Causes.—As has already been stated under their respective headings, physical injury and emotional shock or fright are frequent causes of both hysteria and neurasthenia. This is so common that when the symptoms of these conditions are so caused, they have been termed "a traumatic neurosis."

At one time these cases were termed "spinal concussion," "railway spine," "railway brain," "spinal irritation," etc. These terms are not used now.

Either one of the causes mentioned or a combination of both may cause the condition. Even, however, when the traumatism has caused physical injury, the element of fright must be considered. When this is prominent a very slight physical injury may cause the symptoms.

Symptoms.—The symptoms may either appear immediately after the accident or may not develop for days or weeks afterward. In some cases after the accident there may be vomiting or spitting up of blood-stained fluid. This, unless there is injury of the lungs, comes from the mouth and throat, and is characterized by a small number of red blood cells and by the free mixture of mucous epithelium and bacteria which are collected in the buccal and pharyngeal mucous membrane (Strümpell). Women may menstruate after the shock; in others the patient may be in a dazed condition for a time, or have an emotional outburst.

The symptoms are either those of hysteria or neurasthenia, or as is often the case, a combination of the two.

Hysterical tremor and paralysis, both motor and sensory, are probably more common in traumatic cases than those due to other causes. In cases in which there has been injury, as a blow or twist of the spinal column, symptoms referable to that condition may be present, in addition to the purely nervous symptoms. These are pain in the back increased by movement; there is usually rigidity of the back muscles increased by bending the spine in any direction. The pain may extend into the legs or around into the sides. Marked tenderness is found. Owing to the pain caused, walking is interfered with. This condition is known as "traumatic lumbago," and is due to a sprain of the intervertebral joints. Care must be taken not to confound it with tenderness ordinarily present in hysteria and neurasthenia. In this muscular rigidity and pain on motion are not marked.

A test which may be employed to determine the genuineness of pain and tenderness is the so-called "Mannkopf's sign." This consists in pressing over the painful spot, during which the pulse rate should increase. This is valuable if positive, but a negative result does not necessarily prove that there is no tenderness.

Diagnosis.—As these cases are so often the cause of litigation, the diagnosis is important. In the first place, it must be decided if the patient has organic disease of the nervous system, as many of these may be excited by trauma. Except when due to hemorrhage or inflammation, the symptoms of organic disease are not apt to appear acutely. At the same time it must not be forgotten that in addition to a traumatic neurosis, organic disease may develop later.

The following must be borne in mind:

Hematomyelia
Acute transverse myelitis
Hemorrhage from the middle meningeal artery

Meningitis
Multiple sclerosis
Paralysis agitans
Brain tumor
Pachymeningitis, cerebral or spinal
Syringomyelia
Epilepsy
Paresis
Tabes dorsalis.

The methods of distinguishing functional from organic paralysis are given on page 785.

It must be borne in mind that atrophy, except a moderate amount from disuse, and changes in the electrical reactions do not occur in functional palsies.

In *meningitis,* if basal, there will be cranial nerve palsies, and examination of the cerebrospinal fluid will show evidences of it (p. 679).

Epilepsy may be excited by a fright or trauma in one predisposed. The differential points between the epileptic and hysterical attack are given on page 771.

Paresis and tabes dorsalis cannot be caused by trauma unless the patient has had syphilis. It is possible, however, that a blow on the head or back might excite them or aggravate the symptoms if already present.

The differential points of the above conditions are given under their respective headings.

Traumatic lumbago of long standing must be distinguished from:

Caries of the vertebra
Rhizomelic spondylosis.

Either of the above may arise from a blow on the back. X-ray examination will usually show if there is disease of either the bone or joints.

Having decided that no organic disease is present, it may be necessary to determine if the patient is **malingering**. This may be difficult, although as a matter of fact, it is not very common.

The fact that the patient may exaggerate his symptoms is not proof, as that is characteristic of all cases of hysteria.

The same holds good in showing that while sensory paralysis is apparent, it can be proved that the patient does perceive. This is so in all cases of hysterical paralysis. By means of the stereoscope, for instance, it can be shown that a patient apparently blind in one eye can see with both.

It must also be remembered that these patients are unusually liable to receive suggestions. Hence the examiner may cause the appearance of new symptoms if he is not cautious. Leading questions must be avoided.

Certain symptoms cannot be simulated, as contractures, tremors, contraction of the visual fields, especially if tubular, fibrillary tremors, vasomotor phenomena, persistent tachycardia, bradycardia, exaggeration of the tendon reflexes, especially if they are about the same intensity at different examinations. Sensory paralysis possibly may be.

It is important in testing for it that the patient does not know what you are going to do. Not many can experience a sudden prick of a pin that they are not prepared for without showing some evidence that they feel it.

If the boundaries of the anesthetic area are typical of the types usually found in hysteria, and are more or less constant within the same boundary, it would be against simulation.

Pain is also a subjective symptom, and one that both the hysteric and neurasthenic is apt to exaggerate. The existence of Mannkopf's sign (p. 793) is of service if present. Hysterical joints may present difficulty, and the diagnosis depends upon the absence of signs of organic disease in the joint, as shown by x-ray and other examination, plus other symptoms of hysteria.

The diagnosis will often depend upon the general demeanor of the patient. his circumstances in life, careful watching, and several examinations.

(*d*) *Psychasthenia*

Psychasthenia is the term applied to a group of symptoms which at one time was classified as belonging to neurasthenia. In fact the symptoms are those of neurasthenia plus various obsessions, morbid fears, doubts, compulsions, impulsions, enfeebled will power, and uncontrollable movements of an epileptiform nature.

Special Symptoms.—The *obsessions or fixed ideas* consist of ideas which in themselves are trivial, but of which the patient cannot rid himself and which, more or less, dominate his conduct. They are often unpleasant and relate to possible injury that may occur to either the patient or his relatives.

Morbid fears consist of a dread of doing certain common acts, such as a fear of going into open places (agoraphobia), fear of a closed place or crowd (claustrophobia), fear of personal defilement (mysophobia), fear of storms or lightning (astraphobia), etc. Attempts to perform any of the dreaded actions cause feelings of suffocation, vertigo, headache, and nervousness.

Morbid impulses consist of impulses which spring suddenly into consciousness; and acts follow at once which the patient is unable to resist. They may be trivial acts, as touching every lamppost as the patient passes it, to avoid stepping on a door sill, etc., or they may be harmful ones, as murder, arson, or theft.

The desire to repeat certain words over and over is known as *onomato-*

mania, that of counting a certain number of times before performing an action, as *arithmomania. Kleptomania, pyromania, homicidal and suicidal impulses, and dipsomania,* may be due to this condition. ***Tic*** (p. 764) is a form of morbid impulse.

Doubts, or doubting mania, is a condition in which the patient is never sure he has performed any certain act correctly and may, for instance, retrace his steps a number of times to see if he has locked a door.

Conditions to Be Differentiated from Psychasthenia

Psychasthenia must be distinguished from:

Neurasthenia
Hysteria
Epilepsy
Insanity.

NEURASTHENIA.

In true neurasthenia obsessions are absent.

HYSTERIA.

The distinguishing features of hysteria are given on page 774.

EPILEPSY.

The attacks of clouding of consciousness, vertigo, palpitation of the heart, etc., sometimes known as *psycholepsy,* may be mistaken for epilepsy. Consciousness is not completely lost and the other symptoms detailed above are characteristic.

INSANITY.

The mental symptoms of psychasthenia differ from those of insanity in that their absurdity is recognized. Such cases may, however, cross the line and later become insane.

(*e*) *Anxiety Neurosis*

The anxiety neurosis is related to the above. It has been defined as an intense anxiety or fear that evil of some nature is going to occur to self or friends. There may be some real cause for anxiety, but that manifested is out of proportion to the seriousness of the cause. It is a relatively excessive fear.

Mental Symptoms.—The mental symptoms consist of a mixture of dread, panic, terror, anguish, and apprehension.

Physical Symptoms.—The physical symptoms may be pseudo-angina, a sense of oppression, increase of urine and sweat, diarrhea, suppression of the saliva and gastric juice, and paresthesias. In pure form it is thought in many cases to be due to sexual repression.

Diagnosis.—It may occur as a symptom of melancholia, dementia precox, neurasthenia, and psychasthenia; the diagnosis as to which depends on the accompanying symptoms.

(*f*) *Occupation Neuroses*

(*Professional Neuroses*)

An occupation neurosis is a condition occurring in those who are required to make a constant repetition of fine coördinated movements, in which they lose the power of making these movements while all others can be performed as usual. Less frequently the movements may be simple and coarse. It is an exhaustion neurosis.

Occurrence.—It is more apt to occur in neurotic individuals, and symptoms of neurasthenia are frequently associated and may precede its development.

Writers' cramp, or writers' palsy, is the most common and may be taken as a type. Men are more commonly affected than women.

Forms.—Three forms have been described, i. e., *paralytic, spasmodic,* and *tremulous.* They are rarely sharply defined, there being more or less overlapping of the symptoms of each type.

Usually the first symptom noticed is that after writing for a time a dull ache is felt in the wrist and metacarpal joints, and a sense of fatigue is felt. After a rest this passes away to return again when writing is attempted. Gradually the ache may extend up the arm as far as the shoulder, and other symptoms appear.

In the paralytic form the fingers become unable to hold the pen and it drops from the fingers.

In the spasmodic form the pen is grasped more tightly and either cannot be moved at all, or the pen is dug into the paper.

In the tremulous type there is marked tremor when writing is attempted or the pen grasped.

The spasmodic form is the most common. The spasm is tonic. This type is the usual one caused by other occupations. There may be more or less constant pain and aching in the arm, and sometimes feelings of either numbness, tingling, pressure, or constriction. The aching may follow the course of the median and radial nerves. The cervical vertebra are sometimes tender, and headache in the parietal region of the opposite side has been noted. Rarely is there a quantitative increase to the constant and induced currents.

Other occupations liable to this disorder are telegraphers, typewriters, piano players, seamstresses, drummers, weavers, money counters, compositors, blacksmiths, and others.

Diagnosis.—The history and mode of development should make the diagnosis easy.

It may, however, be mistaken for *neuritis,* and in some cases this may coexist. If neuritis is present there will be tenderness over the nerve trunks, vasomotor symptoms, as congestion; muscular atrophy and weakness in performing all movements and not only some particular ones.

It may also be well to remember that difficulty in writing may be an early symptom in *paresis, paralysis agitans, multiple sclerosis, chorea,* and *progressive muscular atrophy.* Careful examination will disclose other characteristic symptoms of these diseases.

(*g*) *Tics*

These have been described on page 764.

6. Disorders of Sleep

These include (*a*) *insomnia,* (*b*) *morbid somnolence,* (*c*) *morbid dreaming,* and (*d*) *somnambulism.*

(*a*) *Insomnia*

By this term we designate a condition characterized either by habitual incomplete sleep or by periods of entire absence of sleep.

All persons do not require the same amount of sleep. The average amount required by an adult is eight hours, but many people do well with six or less, while others require more.

Mild degrees of insomnia may be a family trait that causes nervousness, irritability, and inability to perform sustained mental or physical labor.

Insomnia is a symptom of many different conditions. It is frequently one of the early symptoms of neurasthenia and of the acute psychoses, as mania or melancholia.

Causes.—Prolonged mental effort and strain, anxiety of mind and worry, visceral diseases, especially those of the heart and arteries, and the abuse of alcohol, tea, coffee, tobacco, and other narcotics, may all be causes.

(*b*) *Morbid Somnolence or Drowsiness*

This may be a symptom of a number of different conditions. It is frequently an early symptom of cerebral syphilis, and is then usually associated with severe headache. It may also occur in brain tumor and be due to arterial degeneration and various forms of toxemia, especially in uremia, diabetes, and constipation. It may follow an epileptic convulsion, especially if there have been a number of them within a short period (status epilepticus), and may be hysterical.

A condition known as *narcolepsy* is characterized by an imperative desire for sleep occurring at intervals. In these attacks it only lasts a few minutes and resembles natural sleep, but wherever the patient is, no matter what he may be doing, he goes to sleep for this brief period.

Cause.—It is a rare neurosis and the cause is unknown.

Conditions to Be Differentiated from Morbid Somnolence

It must not be confounded with the following diseases:

Petit mal type of epilepsy
Trance and catalepsy
Sleeping sickness of Africa.

PETIT MAL TYPE OF EPILEPSY.

In these attacks the patient becomes unconscious for a brief interval but there is no preceding desire for sleep.

TRANCE AND CATALEPSY.

Trance and catalepsy have been described on pages 776-777.

THE SLEEPING SICKNESS OF AFRICA.

This condition is characterized by apathy and somnolence and is due to the presence in the blood of a form of Trypanosoma. The disease is fatal and does not occur outside of a certain territory in Africa.

(*c*) *Morbid Dreaming*

Symptoms.—The important manifestations of this are nightmare and pavor nocturnus, or night terrors. Neurasthenics frequently suffer from disagreeable and horrible dreams.

Nightmare is usually due to either digestive disturbance (overeating), cardiac disease, or prolonged worry and excitement. Those who are neurotic are especially liable to it.

Pavor nocturnus is peculiar to children. The child awakens, screaming with fright, and may be in a highly nervous and agitated state for some time afterward. It may be due to various reflex irritations, hereditary syphilis, and may be a manifestation of nocturnal epilepsy.

(*d*) *Somnambulism*

Somnambulism is a condition allied to the hypnotic state. During the state the patient can avoid obstacles and perform ordinary acts automatically. After awaking, nothing is remembered of what occurred during the attack.

The attacks may be periodical.

Occurrence.—It usually occurs in neuropathic persons.

Causes.—The exciting causes may be overeating, mental strain or excitement. A similar condition may follow the hysterical convulsion.

(*e*) *Somnolentia or Sleep Drunkenness*

Somnolentia or sleep drunkenness is a condition of incomplete sleep, in which a part of the faculties is abnormally excited while the remainder is buried in repose. The patient is excited and may be violent. There is a feeling of impending danger which may be the cause of assaults.

Differentiation.—It may be confounded with the MANIACAL CONDITION THAT SOMETIMES FOLLOWS AN EPILEPTIC SEIZURE. A history of such attacks in the past will point to epilepsy as the cause. It must not be forgotten that some sufferers from epilepsy have their attacks only at night and while asleep. In such cases they should be carefully watched for the occurrence of convulsions.

K. Vasomotor Neuroses and Trophoneuroses

In connection with vasomotor neuroses the reader should also consult pages 593-597.

Acroparesthesia (p. 576) is thought to be in many cases due to vasomotor spasm—in fact, is an abortive form of Raynaud's disease.

1. Raynaud's Disease

Raynaud's disease, also known as *symmetrical gangrene, local asphyxia* and *dead fingers,* has been defined by Osler to be "A vascular change, without organic disease of the vessels, chiefly seen in the extremities, but also occurring in the internal parts, in which a persistent ischemia or a passive hyperemia leads to disturbance of function or to loss of vitality with necrosis of the parts."

Occurrence.—In its typical form it is not a common disease, and develops in the majority of instances in females in early life (between ten and thirty years). It may occur in several members of a family.

Causes.—Neurotic and hysterical persons are prone to the disease and shock or fright has excited the symptoms. Exposure to cold and damp and menstrual disturbances have been assigned as causes. In most instances no etiological factor can be determined.

Various Forms.—Osler considers *mild, moderate and severe types.*

Mild Form.—He considers as suffering from a mild form, those who after suffering for years from cold hands and feet begin to have tingling in the fingers and toes, and after exposure to cold, the feet and hands become blue. After exposure to heat they throb and ache, get hyperemic and feel tense and swollen; and it may take several hours for them to become normal. These are the symptoms of what has been regarded as chilblains. Many cases may go no further than this, but, now and then, one who possibly has been exposed to cold longer than usual may notice that the cyanosis persists longer than usual, this being followed by areas of superficial necrosis over the knuckles or in the tips of the fingers. As the weather gets warmer improvement occurs until cold weather returns, when the condition returns. Hands and feet that are permanently either cyanotic or hyperemic and are swollen, cold and clammy, are probably also mild types of the disease (See also Acroparesthesia, p. 576).

Moderate Form.—Cases of more pronounced, but of moderate severity usually begin with numbness and tingling of either one or two or more of the fingers. Later it will be noticed that they are white and cold (local syncope), especially after exposure to cold, and remain so for a few hours, after which they become cyanotic (local asphyxia). As reaction occurs they become red and warm. During the periods of syncope and asphyxia the pulsation of the radial or dorsalis pedis arteries, according to the parts affected, may be feeble. Between the attacks the pulsation is normal. Such attacks may recur at intervals without further symptoms, or shortly after the first attack it will be noticed that the affected parts remain permanently blue or asphyxiated, and the pain is very severe. Gangrene of one or more phalanges may eventually occur, a line of demarcation forming, and after a few weeks they slough off and healing occurs. No further attack may occur, or others follow at intervals of several months and finally recovery ensues.

Severe Form.—In the severe forms gangrene occurs early; the pain is intense, and in addition to the phalanges, the ears or tip of the nose may be affected. Rarely a foot or hand may be lost. In addition the patient may have either urticaria, purpura, hemoglobinuria, attacks of severe abdominal colic, epileptic convulsions, or transient hemiplegia. Death may occur in such cases.

Symptoms in Typical Cases.—The sequence of symptoms in typical cases is pallor and coldness or local syncope, local cyanosis or asphyxia, local hyperemia, local necrosis.

Cases have been described in which cyanosis is the first symptom. It is not paroxysmal, but continuous. Associated with it is diminution of sensation and a lowered surface temperature. Gangrene occurs ultimately. Such cases have been termed local apnea or acrocyanosis.

Conditions to Be Differentiated from Raynaud's Disease

In making a diagnosis of Raynaud's disease, it must be remembered that somewhat similar symptoms may occur in certain organic diseases of the nervous system, viz., NEURITIS, SYRINGOMYELIA (*Morvan's disease*), HEMIPLEGIA DUE TO APOPLEXY, TABES DORSALIS AND MYELITIS. In such cases characteristic symptoms of the respective diseases will be found. In addition the symptoms usually do not follow the characteristic sequence seen in Raynaud's disease, a condition of cyanosis with possible local necrosis or ulceration being the rule, which in syringomyelia and tabes especially occurs without pain. It must be remembered that transient palsies may be due to *spasm of the arteries* in either the brain or cord (pp. 713, 807), and in connection with MIGRAINE (p. 772). In such cases the characteristic vasomotor symptoms of Raynaud's disease are absent, although in migraine there is usually pallor of the face. Areas of necrosis of the skin may occur, as sequelae of the acute *infectious fevers*, especially in MEASLES, TYPHOID FEVER, TYPHUS FEVER, PNEUMONIA AND MALARIA.

DIABETES AND OBLITERATIVE ARTERITIS may be the cause of similar symptoms. In the former the urine will show the presence of sugar; in the latter the patients will likely be advanced in years and pulsation will not be felt in posterior tibials and dorsal arteries of the feet. The condition is not as likely to be symmetrical.

A condition which occurs usually in Russian Jews of middle age may be mistaken. It has been shown by Buerger to be due to an arteritis with subsequent thrombosis, and is termed by him THROMBO-ANGIITIS OBLITERANS. It involves the feet and usually affects one first, although the other may be affected later. Pain is first felt, often involving the entire limb, with tenderness of the calf muscles. The great toe usually becomes a dusky red hue, although if the limb is elevated it may be pale and cold. The pain is often relieved by allowing the foot to hang down. Pulsation is absent in the posterior tibial and dorsalis pedis arteries and ulceration eventually occurs. Intermittent claudication may coexist.

Areas of skin necrosis have occurred in *hysteria*. Such cases are probably due to simulation, the patient causing the areas by the use of some irritant or other means.

A CERVICAL RIB may cause symptoms simulating Raynaud's disease. This will be shown by x-ray examination. Some cases may be due to DISEASE OF THE DUCTLESS GLANDS, as the THYROID.

Raynaud's disease must also be distinguished from:

Erythromelalgia
Scleroderma
Rheumatoid arthritis.

ERYTHROMELALGIA—SCLERODERMA.

The symptoms of the first two are detailed below, where the points of difference will be readily seen (See also table on p. 811). Raynaud's disease may be associated with scleroderma.

RHEUMATOID ARTHRITIS.

It may also occur in cases of rheumatoid arthritis, but independently of this, recurring attacks of Raynaud's disease sometimes cause periarticular thickening and ankylosis of the terminal joints. Effusion has also been reported, being present in the larger joints. X-ray examination will reveal the characteristic changes in the joints of rheumatoid arthritis, the pain will be greater and it will, together with the symptoms of joint disease, precede the appearance of the symptoms of Raynaud's disease.

2. Erythromelalgia

Erythromelalgia is a "chronic disease in which a part or parts of the body—usually one or more of the extremities—suffer with pain, flushing, and local fever, made far worse if the part hangs down" (Weir Mitchell).

Occurrence.—It is a rare condition, and occurs most frequently in men between the twentieth and fortieth year.

Causes.—Puberty, the climacteric, menstrual disturbances and infections, as gonorrhea, syphilis, and rheumatic fever, have been given as *predisposing causes.* Exposure to cold and damp, overexertion or prolonged use of the legs and injury to the leg, may be *exciting causes.*

Symptoms.—The symptoms are redness, severe pain and swelling of the foot, which does not pit on pressure, and sometimes of the leg in addition, which are made worse either when the foot hangs down, is stood upon, or is exposed to heat. The surface temperature is increased, and sweating may occur. When the limb is elevated the symptoms practically disappear.

Cassirer has described a form in which the symptoms are localized to a definite nerve territory without symptoms of neuritis being present. Thickening of the skin, pigmentation and changes in the nails may occur as may also some atrophy of the muscles due to disuse. While the feet are usually affected, the hands may be either in association or independently.

Conditions to Be Differentiated from Erythromelalgia

Somewhat similar symptoms may occur in organic diseases of the brain, spinal cord, and peripheral nerves, viz., HEMIPLEGIA FOLLOWING APOPLEXY, TABES DORSALIS, MULTIPLE SCLEROSIS AND MYELITIS. In such cases the condition is due to an organic lesion of the vasomotor centers in the brain and cord and not to functional disturbance. As has been stated, in similar conditions symptoms simulating RAYNAUD'S DISEASE may also occur (p. 801). The diagnosis depends upon finding the characteristic symptoms of the organic diseases mentioned.

Similar symptoms may also occur in NEURITIS, and Osler mentions two cases in which they occurred in connection with ARTHRITIS AND ANKYLOSIS OF THE SHOULDER JOINT, which was followed by pain in the arm in the course of the nerve trunks. In neuritis in addition to the pain there will be tenderness over the nerves.

CERVICAL RIB must be thought of if the hands alone are affected, and TUMOR INVOLVING THE CAUDA EQUINA may be a cause.

The condition described on page 802 as THROMBO-ANGIITIS OBLITERANS resembles somewhat erythromelalgia. In the former redness may occur when the foot hangs down but the pain is lessened; ulceration and necrosis eventually occur, and pulsation is lost in the arteries of the leg. ENDARTERITIS OBLITERANS may cause somewhat similar symptoms (See Intermittent Claudication).

FLAT FEET should be considered; however, the feet, when standing or walking, do not have the characteristic vasomotor symptoms and the condition is apparent on observation.

In pure form, erythromelalgia must be distinguished from RAYNAUD'S DISEASE and SCLERODERMA. The differential points are detailed in the description of these diseases. They will also be found in the table on page 811. It must be remembered that the features of each may sometimes occur in the same patient.

3. Scleroderma

Scleroderma is a disease in which there is either a diffuse or localized atrophy, induration, and either excessive pigmentation or lack of pigmentation of the skin and subcutaneous tissue, associated with vasomotor symptoms.

Occurrence.—It is a rare condition; it is most common in young adults of the female sex, but may occur at any age.

Etiology.—The acute infectious fevers, acute otitis media, tuberculosis, syphilis, tonsillitis, menstrual disturbances, exposure to cold, emotional disturbances, trauma, neuropathic disposition, migraine, and alcoholism, have been asserted to have etiological significance. In many cases no cause can be assigned. It has occurred associated with exophthalmic goiter.

Modes of Onset.—There are three modes of onset—the *atrophic,* the *edematous* and the *erythematous.* In many cases these symptoms are preceded by rheumatic pains in the limbs and general nervousness. There may be apparent inflammation of a joint or joints, the latter becoming stiff and sore. Such attacks may occur at intervals for several years before the skin becomes affected. As the skin becomes involved a feeling of stiffness is usually first complained of. This is most marked in the neck, shoulders, arms, face and scalp. The skin in these regions will be found to have lost its elasticity. It gradually becomes hard, feels leathery to the touch and in areas there may be a deficiency of pigment, while in others the skin is of a yellowish or brownish color. As the disease progresses the skin becomes thin, smooth, glossy, immovable, and apparently fixed to the bones.

In the face the nose becomes small and the skin about the mouth is retracted so that the teeth may show, the appearance being very much like that of a mummy. Muscular atrophy may occur and kyphosis in either the cervical or thoracic region may develop. Shrinking of the skin of the hands may cause deformities of the fingers, as contractures, ankylosis, and luxations of the phalangeal joints. Ankylosis of the large joints may also occur.

Associated with this, ulcerations occur about the finger nails and phalangeal joints. This condition has been termed *sclerodactylia.* Atrophy of the bones may also occur. In this stage there is usually no pain, but a feeling of stiffness and tension is complained of; the sexual functions are lost; elevation of temperature may occur. These symptoms may be preceded by an *edematous stage,* which may develop acutely. Swelling occurs in the face, breast and arms, less frequently the trunk and legs. This swelling is hard, smooth, and does not pit on pressure. The color of the skin may be either normal or alternately red and mottled, later showing areas in which there is either deficient or excessive pigment. As atrophy of the skin occurs, small areas develop which seem to be sunken below the surface of the surrounding skin, or bands may develop; these are usually paler than the surrounding parts. Eventually the changes detailed above occur. In this stage apparent retrogression and improvement may occur for a time.

The *erythematous onset* is of two types. There may be either a diffuse erythema and swelling involving the face or parts of the trunk, or symptoms resembling those of Raynaud's disease may affect the hands and feet. These symptoms may occur in association with those mentioned above.

Localized scleroderma or morphea occurs as patches or bands in which similar changes in the skin occur, as in diffuse scleroderma. These may follow the course of certain of the peripheral nerves, either cranial or spinal. The patches are white or yellowish in color and are surrounded by a zone which may be either yellow, brown, bluish, gray or lilac in color. The transparency of the affected area has caused it to be compared to bacon rind.

Conditions to Be Differentiated from Scleroderma

The diagnosis after the peculiar changes in the skin have occurred is not difficult. In either of the early stages when in association or when the localized forms only are present, there may be some difficulty.

Therefore in the **edematous stage** the distinction may have to be made from:

Angioneurotic edema
Myxedema
Dermatomyositis
Leprosy.

ANGIONEUROTIC EDEMA.

Angioneurotic edema may be mistaken for the edematous form. The differences will be seen by referring to pages 809, 811.

MYXEDEMA.

In myxedema there is neither the hardness of the skin and peculiar anomalies of pigmentation nor is it marked on the chest as it is found in scleroderma. The mentality is sluggish, the thyroid atrophied, the temperature subnormal, and vasomotor symptoms are absent.

DERMATOMYOSITIS.

In dermatomyositis there is marked pain and tenderness on pressure over the affected area; the skin is red and resembles the appearance either of erythema, eczema, urticaria or erysipelas. The peculiar bands and patches of indurated and immovable skin do not occur.

LEPROSY.

Scleroderma in the erythematous and edematous stages may simulate leprosy. In the former atrophy, induration, and changes in the color of the skin, soon develop—which is not the case in the latter.

When ankylosis of the joints occurs the distinction may have to be made from:

Rheumatoid arthritis.

RHEUMATOID ARTHRITIS.

In rheumatoid arthritis, thinning and glossiness of the skin may occur. It does not develop the hardness and immovability found in scleroderma, nor is the skin of the face affected.

The **erythematous type** must be distinguished from:

Raynaud's disease
Erythema nodosum.

RAYNAUD'S DISEASE.

As has been stated, Raynaud's disease frequently occurs in association with scleroderma. The question to decide is whether the vasomotor symptoms are the primary condition or a complication. Cases in which there have been a number of attacks of the Raynaud syndrome may eventually develop stiffness of the fingers and some hardness of the skin, but it progresses no further. These are evidently true cases of Raynaud's disease. Others, however, may develop the classical symptoms of Raynaud's without the occurrence of gangrene. Gradually these are followed by the typical symptoms of diffuse scleroderma. Such cases are probably scleroderma with vasomotor symptoms.

ERYTHEMA NODOSUM.

Erythema nodosum is a disease usually of comparatively brief duration with acute onset and constitutional symptoms. The skin is not hard and can be pinched up in the fingers.

The **atrophic type** with marked pigmentation may stimulate:

Addison's disease.

ADDISON'S DISEASE.

In this, however, there is very low blood pressure, rapid exhaustion and cachexia, gastric and intestinal achylia, and absence of the peculiar skin changes.

Sclerodactylia, if accompanied by ulceration, may be mistaken for:

Syringomyelia.

SYRINGOMYELIA.

In this, however, there are not the characteristic skin symptoms, while dissociation of sensation, spastic paraplegia, and muscular atrophy, are more or less developed.

The **localized form,** if confined to the face, may be mistaken for:

Facial hemiatrophy.

FACIAL HEMIATROPHY.

In this the peculiar condition of the skin is not found, and the bones and skin atrophy synchronously.

4. Intermittent Claudication

Dyskinesia angiosclerotica intermittens, also known as *dysbasia angiosclerotica intermittens, intermittent claudication,* and *intermittent lameness,* is a condition the symptoms of which depend upon arterial degeneration plus spasm of the affected vessels. It is therefore not a true neurosis, but may well be described here as it may be confounded with vasomotor neuroses.

Occurrence.—It is most common in men past middle life, but may rarely occur in young persons.

Causes.—Prolonged exposure of the limbs to cold and moisture, excessive use of tobacco or alcohol, and, rarely, syphilis, have been assigned as causes.

The usual type of case is due to arterial sclerosis affecting the arteries of the legs.

Symptoms.—The symptoms are as follows: the patient, while at rest or while walking a short distance, has normal movement and strength of the legs. After walking for from a few to fifteen minutes, feelings of weight, numbness, and possibly cramp, are felt in the limbs.

The sensations increase to actual pain and weakness so that the patient must sit down and rest. After a rest he can go on as before for a short distance, when the symptoms will be repeated as before.

In such cases the pulse in the dorsalis pedis and posterior tibial arteries will be found either absent or very weak. During the interval it may sometimes be weakly felt, while during the attack it is absent. During the attack and sometimes in the interval the foot of the affected side is paler and colder than normal. One limb only may be affected, or rarely, the arms may be (dyspraxia angiosclerotica intermittens).

The symptoms are probably due to a blood supply which, owing to the rigid and narrowed arteries, cannot be increased to meet the requirements of the muscles when in motion. In addition to this, spasm of the artery may also occur.

Spasms of the cerebral arteries causing apoplectic symptoms and in migraine have been previously described (pp. 713, 772).

They may also affect spinal arteries, causing transient attacks of paraplegia, with possibly paralysis of the sphincters and loss of sensation. Intermittent claudication, as described above, may have to be distinguished from this form.

In the spinal type there will be absolute paralysis of both limbs for the time being, which does not disappear after rest to reappear after exertion. The arterial pulsation can usually be felt and the sphincters are involved.

Additional Conditions to Be Differentiated from Intermittent Claudication

In addition intermittent claudication must be distinguished from:

Myasthenia gravis
Rheumatism
Thrombo-angiitis obliterans
Raynaud's disease
Neuritis.

MYASTHENIA GRAVIS.

In myasthenia gravis all the muscles become weak after they are used. There are no sensory symptoms. Arterial pulsation is good and the myasthenic reaction is present.

RHEUMATISM.

Rheumatism of either the muscles or joints may be mistaken. In this, however, the patient is apt to feel better after moving about and the sequence of motion after rest and paralysis after motion does not occur. Arterial pulsation is present.

THROMBO-ANGIITIS OBLITERANS.

In the early stages of thrombo-angiitis obliterans (p. 802), or obliterans arteritis, there may be similar symptoms, and then distinction may be impossible. In a short time, however, constant pain, redness of the toes, and necrosis will develop.

RAYNAUD'S DISEASE.

The pallor of the foot sometimes present may be mistaken for that of Raynaud's disease. The pallor is not followed by hyperemia (unless the condition above mentioned is developing), when it will not be paroxysmal. In Raynaud's disease there is no weakness of the limbs or cramplike pains.

NEURITIS.

Neuritis, especially of the sciatic nerve, may be mistaken, as pain and consequent difficulty in walking are aggravated by motion in that condition. There will, however, be tenderness along the course of the affected nerve aggravated by flexing the thigh on the body; the Achilles jerk may be absent; pain is usually felt when the limb is at rest and arterial pulsation is present.

5. Angioneurotic Edema

Angioneurotic edema, or *Quincke's disease, giant urticaria, intermittent edema*, and *urticaria edematosa,* is a vasomotor neurosis in which localized swellings of the skin, subcutaneous tissues and mucous membranes occur spontaneously and last from a few minutes to several hours.

Causes.—In most cases no exciting cause can be discovered, but in some either errors in diet, nervous strain, rheumatism, malaria, menstrual disturbances or trauma have etiological significance.

Many of the patients are neurotic and are neurasthenic or have migraine or other neurosis. It is frequently hereditary and recurrent.

Symptoms.—The symptoms may be mild, moderate, or severe. In the mild cases, after possibly a feeling of itching or burning, a localized swelling rapidly develops in either the forehead, side of the face or back of the hand, eyelid, or lip. This lasts a few hours and then disappears. Similar attacks may occur at intervals of a few months or a year. The health otherwise is not disturbed.

In more severe cases the swelling is greater, so as to close the eyes or interfere with eating, and lasts longer—in some cases being continuous. The mucous membranes may be affected, and hemorrhagic edema of the walls of the stomach and intestines may cause colicky pains, vomiting, and diarrhea, which may be bloody.

In the severe cases, in addition to swellings of the aggravated type elsewhere, edema of the throat and glottis may occur to such an extent as to threaten life. In fact death has been so caused.

In some locations there may be pitting on pressure, in others not. The color may be either bluish white, opaque white, or translucent and waxen. There may be either no sensation or itching and burning. Albuminuria rarely occurs, and there may be headache, somnolence, vertigo, and mental depression.

Conditions to Be Differentiated from Angioneurotic Edema

Angioneurotic edema must be distinguished from:

Hysteria
Milroy's edema
Severe urticaria
Local thrombosis
Scleroderma
Appendicitis.

HYSTERIA.

The edema of hysteria has been described on page 781. It is not recurrent or hereditary.

MILROY'S EDEMA.

Milroy's edema, or hereditary edema of the legs, is a rare hereditary disease in which the swelling is confined to the legs and is permanent.

SEVERE URTICARIA.

In severe urticaria there is intense itching, and the swelling can be produced by irritating the skin.

LOCAL THROMBOSIS.

Cases in which there are local thrombi in the veins of the legs may be difficult to distinguish. Careful examination and the history and mode of development of the swelling may reveal the true cause.

SCLERODERMA.

In scleroderma in the edematous stage there is swelling which is exceedingly hard, permanent, and possesses other peculiar features detailed on page 805.

APPENDICITIS.

The abdominal pain and vomiting often present may lead to a suspicion of the presence of appendicitis. The occurrence of the swellings and the absence of localized tenderness and abdominal rigidity distinguish the two.

Résumé.—The following table gives the **differential points useful in distinguishing the various vasomotor disturbances:**

	Raynaud's Disease	Erythromelalgia	Acroparesthesia	Acrocyanosis	Thrombo-angiitis Obliterans	Scleroderma	Intermittent Claudication	Angioneurotic Edema
Sex	Usually females	Usually men	Usually women	Most of reported cases have been men	Usually men, (Russian Jews)	Usually women	Usually men	About equal
Onset	Pallor of fingers or toes, usually the fingers	No change unless the part hangs down, then it is red. Usually the feet are affected	Numbness and tingling of hands, often just before rising in the morning	Cyanosis without preceding pallor	May be pallor if foot is elevated, red if it hangs down. Preceded by pain and tenderness	Characteristic changes in the skin either before or shortly after occurrence of vasomotor symptoms	Weakness and pain in limbs after exertion	Periodical development of localized edematous swellings which may pit on pressure
Symptoms	Pallor followed by hyperemia or cyanosis, later necrosis; either no pain or at intervals it may be severe, not affected by position	When hanging down, foot red, arteries pulsate, heat increased. Symptoms disappear when foot is elevated. No necrosis	Above symptoms continue till condition is present in daytime also. Aggravated by using fingers or putting hands in water	Continual cyanosis not relieved by heat or position	Constant pain, sometimes relieved if foot hangs down. Necrosis of toes later	Hardness, contraction and pigmentation of skin. Necrosis of skin over joints sometimes	After rest, strength returns, to be followed by pain and weakness after exertion. Foot sometimes pale	Colicky pains, swellings, which disappear and recur
Conditions of arteries	Pulsation in arteries of arm or leg between attacks	Pulsation for time being may be increased	Pulsate	Pulsate	Pulsation absent in posterior tibial and dorsalis pedes	Pulsation present	Pulsation absent or weak in posterior tibial and dorsalis pedes	Pulsation present
Effect of heat and cold	Aggravated by cold	Aggravated by heat, relieved by cold	Apt to be worse in cold	Worse in cold	May be temporarily relieved by heat	Varies	No effect	No effect
Sensation	Pain during attack. Anesthesia and analgesia in affected parts	Normal except pain when foot hangs down	Not affected, except as mentioned above	May be anesthesia and analgesia	Constant pain. May be sensory loss or diminution in affected limb	No change	No change	No change
Temperature	Affected parts cold. Not affected by position	Affected foot hot when hanging down	Hands may or may not be cold	Parts cold	Affected foot cold, may be warmer when hanging down	No change	Foot may be cold during spell	No change
Parts affected	Usually the fingers, toes, tip of nose and nose more rarely. Usually symmetrical	Feet, rarely hands. Not symmetrical	Usually the hands	Hands or feet and legs	Toes and foot. Not symmetrical. One side may be affected after the other	Face, arms, and chest	One or both legs	Face, lips, hands, mucous membrane
Gangrene	Either fingers, toes, tip of nose or ears. Local, usually symmetrical	No gangrene	None	Gangrene may occur	Toes, possibly foot. Not symmetrical	Superficial gangrene of skin over finger joints. Sometimes Raynaud's disease may be present	Gangrene of foot may occur later	None

Progressive facial hemiatrophy, acromegaly, hypertrophic pulmonary osteo-arthropathy, achondroplasia, and leontiasis ossea, are frequently classified as trophoneuroses.

As regards acromegaly, this is incorrect, as we now know it is due to disease of the pituitary gland. It and the others mentioned are described in other places.

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